

THE
WEST RIDING LUNATIC
ASYLUM

MEDICAL REPORTS.

EDITED BY

J. CRICHTON BROWNE, M.D., F.R.S.E.

AND

HERBERT C. MAJOR, M.D.

VOL. VI.

LONDON:
SMITH, ELDER, & CO., 15 WATERLOO PLACE.
1876.

PRICE 9/8.

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'Of all known substances, nerve is the most changeable, the most impressible, the most readily adaptable to changing combinations of incident forces,—in short, the most easily differentiable and integrable.'

FISKE.



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THE
WEST RIDING LUNATIC
ASYLUM
MEDICAL REPORTS.

VOL. VI.

This volume very rare
C.C.D.

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PREFACE.

THE Sixth Annual Volume of West Riding Asylum Medical Reports contains the records of the most noteworthy observations recently made by the Medical Staff of the Hospital, and a few contributions from labourers in the field of nervous pathology, who, although not officially connected with the Asylum, are interested in the work that is carried on in it.

THE HISTOLOGY OF THE ISLAND OF REIL.

By HERBERT C. MAJOR, M.D.

MEDICAL DIRECTOR WEST RIDING ASYLUM.

THE present Article is intended to form the first of a series having for its object the investigation of the minute structure of the Island of Reil as found in the human brain under varying conditions and in the ape.

Little need here be said with the view of advocating the claims of the Island of Reil, Insula, or Central lobe of the brain, as being worthy of the closest and most careful study on the part of the anatomist; not that it is more important than any other part of the cerebrum, but because, in some respects, it has special features of interest. It is that part of the brain which appears earliest both in the human fœtus and in the animal series. It appears to be peculiar to man and the higher apes; with the exception of the Makis, no indication of its presence being observed in other animals.¹ Situate also as the lobe is, deeply in the cerebral mass, and concealed between the frontal, parietal, and temporo-sphenoidal lobes, it is, in those of the lower animals in which it occurs, peculiarly difficult of access to the experimental investigator. And finally, there would seem to be

¹ Gratiolet et Leuret, 'Anat. Comp. du Syst. Nerveux,' p. 111.

good grounds for believing that this region of the cortex is connected in an especial manner with the exercise of the faculty of language.

The above facts have for the most part long been known; but the directions in which they point with irresistible force, as those in which research should be conducted, have, as I believe, been greatly overlooked, so that facts which ought ere this to have been elicited, and questions which ought to have been set at rest, have still to be worked out and brought to light.

Since the time when Fr. Gennari inaugurated the study of the cerebral cortex (1782), or even when Baillarger published his admirable memoir,¹ which ought to serve in all time as a model of accurate investigation and clear exposition, great additions have doubtless been made to our knowledge of the minute anatomy of the brain. But much remains to be done in many directions, and one of these, as before stated, is, I believe, the Island of Reil.

The ordinary descriptive anatomy of the Insula, the manifold courses taken by the fibres which bring the lobe into relation with other parts of the cerebrum, have been carefully studied and described by Meynert, Luys, L. Clarke, Broadbent,² and others. With regard, however, to the minute anatomy of the cortex of the Insula, while it may be that unwittingly I do injustice to some whose labours have escaped my observation, I am not aware that the part has attracted heretofore special study, with the view of ascertaining its exact structure.

The comparative anatomy of the lobe, again, is touched upon, in the briefest manner, by Gratiolet, who bestows merely a few vague sentences on its characters in the Orang and Chimpanzee;³ and I am not aware that any other author has extended our knowledge in this direction.

And thus it is also with regard to the relative structure of the Insula in the fœtus and young child as compared with the adult; our knowledge is vague, uncertain, incom-

¹ Baillarger, 'Structure de la Couche Corticale.' Paris, 1840.

² See more especially Broadbent, 'The Structure of the Cerebral Hemisphere.'

³ Gratiolet, 'Mémoire sur les Plis Cérébraux de l'Homme et des Primates.'

plete. And lastly, in the consideration of those atrophic changes which, under certain circumstances, induce wasting and destruction of the nervous structure, the condition of the Insula, and the extent to which it is involved, are, I have reason to know, very generally passed over, and often, I cannot doubt, to the loss of important data. Now all these questions are well deserving of careful consideration, and it is to them I desire in this and subsequent papers to direct attention. And it seems natural and advisable that the opening Paper should be devoted to a consideration of the histology of the Insula in the healthy human adult, an accurate knowledge of which is essential to a right judgment on the other points of the enquiry to which I have alluded.

The questions, then, now proposed, and the answers to which it will be my endeavour to supply in the present Article, resolve themselves into the following :—

I. Can any structural peculiarity be detected in the arrangement of the cortical layers of the human Insula, or in the nervous or other elements composing them?

II. Do the gyri which collectively form the Insula, agree with each other in minute structure?

III. Does any structural difference exist, under ordinary conditions, between the right and left Insula?

IV. Does the white matter of the Insula maintain the same plan of union with the cortex, as regards the course of the fibres, as in other convolutions generally?

I. It is not strange that at the time when Fr. Gennari, Vic d'Azyr, Meckel, Cazauveilh, and even Baillarger wrote, considerable differences of opinion should have existed with regard to the layers of the cortex of the brain, and that, consequently, the descriptions given by these authors should have varied. For them, the chief method of investigation consisted in pressing a small portion of brain substance between two pieces of glass, and examining it with the unaided eye, or, at most, very imperfectly magnified. But it is strange that more recent investigators, with all the means for accurate observation at their disposal, should, on a simple matter of observation, fail generally to arrive at a common conclusion. And yet such is the case.

Kolliker¹ distinguishes four cortical layers as constituting the general plan of arrangement. L. Clarke² gives the number as eight; Th. Meynert,³ five; Charcot,⁴ five. Doubtless there are many sources of difficulty and fallacy. Frequently it happens that at one spot the cortical layers appear so mingled and thrown into confusion as to render a candid and truthful enumeration of them almost a matter of impossibility; while in another section, taken only a line from the first, the layers of cells manifest themselves with unmistakable accuracy and precision. But then, such accidental sources of fallacy should disappear before systematic and extended observation, and should not long occasion doubt. And in truth, much of the uncertainty and confusion on the subject, and which are only now beginning to pass away, thanks to the labours of Bets and others, would have been avoided if authors had more frequently delineated the objects which they desired to describe. In so complex a study as that of the structure of the brain, long descriptions, unaided by actual demonstration or by plates, are in reality of little value.

Since, then, there exists this diversity of opinion with regard to the elementary points of cortical structure as usually presented to us, it is necessary that I should in the first place state what, in my opinion, is the general plan and arrangement of the layers in the convolutions of the vertex, so as to be in a position to draw comparisons between such arrangement and that found in the Insula in man and in the apes.

The method of preparation of the brain tissue adopted in making these investigations, as well as the source from which sections of the normal Insula were obtained, should first be stated. With regard to the former point it will be sufficient for me to say that Clarke's method has been followed. The fresh method of preparation which my colleague, Mr. Bevan Lewis,⁵ has done so much to improve and extend, is here, I regret to say, not available, or only to a

¹ 'Histologie Humaine.'

³ 'Stricker's Handbook,' vol. ii.

² 'Proceed. Roy. Soc.,' September 1863.

⁴ 'Progres Medecal,' 1875.

⁵ 'Monthly Micro. Journal,' September 1876.

very slight extent. The disadvantage of the fresh method is that by it the operator does not know *exactly* what he is investigating—cannot, for instance, pick out the fourth cortical layer and examine it, excluding the admixture of other layers. Doubtless this is a difficulty which patience and ingenuity will in time surmount, but at present it is fatal to such an investigation as the comparative structure of the cortical layers.

The preparations which have served as my standards of comparison for the healthy structure of the Insula were provided by the brain of a young man æt. 24, who was accidentally killed when, so far as could be ascertained, in a condition of full health.

In a Thesis presented to the University of Edinburgh (1875), on the ‘Histology of the Brain in Apes,’ I described six cortical layers as being the usual arrangement in the human brain. In the ‘Journal of Mental Science’ for January 1876, in a paper on the brain of the Chacma Baboon, I again showed that in the human subject the six-layer type of the cortex was the usual one. Now this is not the number as given by the majority of histologists, and it is necessary, therefore, that I should explain where it is that we diverge.

An examination of the drawings I have given (Plates I. and II.), and their comparison with that given by Meynert (‘Stricker’s Handbook,’ vol. ii. p. 234), will make the point of divergence clear. It will be at once observed that with regard to the first, second, third, and fourth layers we are at one, but beneath the fourth layer Meynert figures *one* layer, while I give *two*; and hence Meynert describes five layers, while I, following Baillarger, describe and figure *six*. Doubtless, modifications in the general appearance of the layers are frequent. To illustrate this it is only necessary to pass successively in the field of the microscope, under a low power, the cortex of the summit of a gyrus and that in a sulcus, as figured in Plates I. and II. Yet here is no alteration in the six-layer type, but merely a modification of it.

Now the first point I desire to establish is this—that the *plan of arrangement* of the cortical layers in the Island of

Reil differs in no respect from that I have already alluded to as being the ordinary one throughout the cortex. If the layers delineated in Plate I. be compared with those figured by Meynert, it will be seen that, with exception of that which I term the 5th layer, and which Meynert does not notice as distinct from the deepest, the resemblance is nearly complete. The cells may not have the same relative or absolute size—that is a point which will be considered shortly—but their general aspect as seen with a low power of the microscope, and their relative numbers in the several layers, correspond very closely.

With regard to the intimate constitution and appearance which the nerve-cells of the Insula present, as seen under a power of 350 *diameters*, I can observe nothing unusual:—nothing that would seem to imply (as in the case of the so-called giant cells of the vertex) any special and peculiar functions. Even with the highest magnifying power at my command (*one-tenth* objective, Hartnack), I can detect no departure from those characters which are so well recognised.

The size of the nerve-cells of the Insula, as of all other parts of the cerebrum, deserves special attention. I gather from the account of Dr. Lockhart Clarke,¹ that, while he considers the nerve-cells of the Insula to be generally larger than in some other parts, yet that they are not as large as those commonly found in the convolutions of the vertex.² I am quite sure of the general accuracy of the above remark, but would wish to extend the observation and render it more precise by an appeal to actual measurement of the cells of the various layers as taken with the micrometer.

1st layer. The small and for the most part nucleus-like bodies which occur in this layer, I find measure $\cdot 008$ *millimetre*, the occasional nucleated corpuscles being about $\cdot 012$ *millimetre* (Plates I., II., 1 1').

2nd layer. The small pyramidal bodies of the second layer (I., II., 2 2'), average $\cdot 012$ *millimetre*, occasionally reaching $\cdot 016$ *millimetre*.

¹ Maudsley's 'Physiology and Pathology of Mind,' 3rd ed. p. 115.

² This is, I believe, Dr. Clarke's meaning.

3rd layer. The cells of the third layer vary between $\cdot 016$ at the superficial portion of the stratum, to $\cdot 024$ or even $\cdot 028$ *millimetre* (occasionally only) at the deepest part. It will be observed that in this layer, as the cells increase in size, they diminish in number (I., II., 3 3').

4th layer. The small oval or pyriform bodies of this layer, which impart to the stratum so distinctive an appearance, owing to their small size and especially to their great uniformity, measure, with few exceptions, $\cdot 012$ *millimetre*. Occasionally, as in other parts, a large cell measuring $\cdot 02$ or even $\cdot 024$ *millimetre* is seen, but this is rare. The sudden diminution in the size of these bodies as compared with those of the previous layers is very striking, coinciding as the fact does, with the result of simple observation.

5th layer. In this layer, I believe, more than in any other, the cells vary in size, and it is the more difficult to give an approximate average. I think, however, that the dimensions $\cdot 02$ to $\cdot 224$ *millimetre* will include most of the corpuscles. Many, however, are very much smaller, while, on the other hand, some occasionally occur measuring $\cdot 032$ *millimetre* in length (I., II., 5 5').

6th layer. The corpuscles of this, the deepest cortical layer, are for the most part spindle-shaped, more especially in that part of the cortex forming the summit of a gyrus, and hence their length is very disproportionate to their breadth. Average length, $\cdot 02$ to $\cdot 024$ *millimetre*; breadth, $\cdot 008$ *millimetre* (I., II., 6 6').

The above results are those afforded by the healthy brain to which I have before alluded, but they have been supplemented and confirmed by measurements taken in twelve other cases, which, although morbid, afford valuable *confirmatory* evidence. It is hardly necessary to point out the impossibility of giving in any instance the exact dimensions of all the corpuscles, a fair average derived from a large number of observations being the most that can be afforded.

Taking now a section from a convolution of the vertex (frontal region), it will be seen that the estimated dimensions of the cells show a close resemblance as regards absolute as

well as relative size, thus (as before in fractions of a *millimetre*):

1st layer . . .	·008 to ·012	millimetre
2nd layer . . .	·012 „ ·02 (rarely) „	
3rd layer . . .	·02 „ ·028 „	
4th layer . . .	·012 to ·02 (rarely)	millimetre
5th layer . . .	·02 „ ·024 „	
6th layer . . .	·016 „ ·02 „	

Now a comparison of the above figures with those before given as representing the size of the cells in the Insula, might hardly seem to warrant the statement formerly put forward that in the latter situation the cells are smaller than at the vertex. The truth, however, and its explanation, appear to be this:—The *third layer* is that in which the contrast occurs. In the Insula cells are found quite equal in size to those in the corresponding layer at the vertex (I exclude, of course, from consideration the so-called giant cells); but the *majority* are smaller; and hence it is that while in a section taken from the vertex the band of cells forming the third layer stands out from all the others, in the Insula it is much less conspicuous.

The above, then, I apprehend to be the chief feature of distinction; and while at present I would not venture to suggest an inference, there can, I think, be no doubt that the point is an important one. For it must be remembered that it is in the cells of the *third layer* that degenerative changes described by myself and others are most frequently apparent.

II. The next point for consideration is as to whether any structural differences can be detected between the several gyri which collectively form the Insula. To ascertain this, sections were made through the entire Insula at different levels, so that in each section the gyri were viewed side by side, and structure could be compared under exactly similar conditions. The result, however, has been negative; no structural variation being discoverable in the various gyri, even under the severe comparison above indicated. This result is

probably in accordance with what would have been anticipated, but nevertheless it is, I consider, important.

III. In like manner I have failed to establish, after a careful and prolonged search, any structural difference between the right and the left Insula. Doubtless the point is a difficult one to determine with absolute certainty, but at present I am decidedly of opinion that the structure on both sides is identical; and if the opinion is expressed with some confidence, it is only because I know that peculiarity of structure does not readily escape detection when the eye (to put it so) has become perfectly familiar with the intimate structure of the cerebral tissue.

IV. The course of the fibres issuing from the Island of Reil has been studied and described more especially by Clarke, Meynert, Gratiolet, and Broadbent, who have dealt very fully with the subject. The only point I desire to refer to has reference to the course of the fibres as they pass into the cortex of the Insula. It was shown by Baillarger,¹ and again by Broadbent,² that all parts of the cortex do not receive fibres coming directly from the central stem of white matter, and the portions which do not so receive fibres are those at the bottom of the sulci between the convolutions. Baillarger states, with perfect accuracy, that long and numerous fibrils run from the central white stem to the *summits* of the convolutions; that such fibrils become rarer and shorter as the sulcus is approached, and become transverse at the sulcus, where the white matter is almost, as it were, *applied* to the cortex, instead of being fused with it as it is at the summit of the gyrus. Hence it is that, as also remarked by Baillarger, a section of brain (in some of the lower animals more especially) will often show the cortex, owing to a little pressure, actually separating from the white matter at the bottom of a sulcus. Now in the human Insula the same arrangement holds good, the fibres for the most part curving round the cortex at the bottom of the sulci, instead of passing upwards into the grey matter.

With regard to the blood-vessels and neuroglia of the Insula, I have been able to observe no peculiarity calling for special description or remark.

¹ Loc. cit.

² Loc. cit.

Such, then, are the conclusions to which I have arrived, and with their brief recapitulation will close this the first part of the present enquiry.

In reply to the four questions proposed at the outset of this Article, I would answer as follows:—

1. The cortical layers of the Insula agree in number, order, and general arrangement with those of the vertex, but the cells of the *third* layer are in the Insula generally smaller than at the vertex. The vessel and neuroglia present no peculiarity.

2. The various gyri forming the Insula present similar structure.

3. No difference of structure can be detected in the right as compared with the left Insula.

4. The method of union of the white matter with the cortex is in the Insula similar to that observed in other lobes.

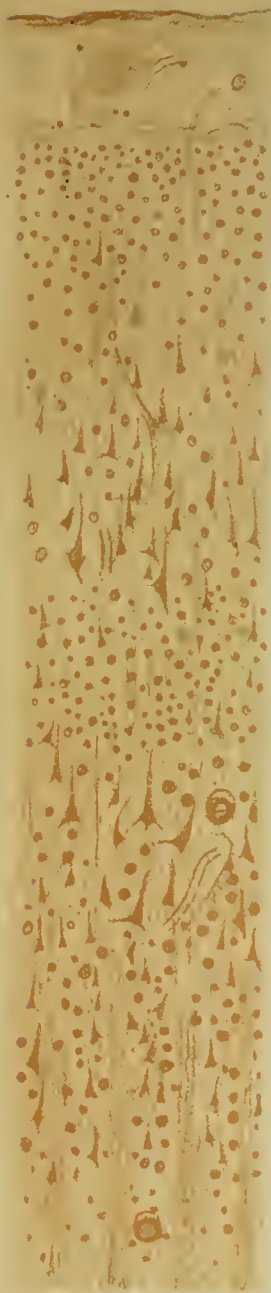
EXPLANATION OF PLATES.

Plate I.—Section through a gyrus of the Island of Reil, showing the cortex of the *summit* of the gyrus (healthy).

Plate II.—Section through the cortex of the Island of Reil at the *bottom of a sulcus* (morbid).

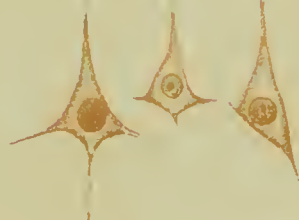
In both:—1, 2, 3, 4, 5, 6, indicate the cortical layers magnified 50 diameters.

M M Medulla. 1', 2', 3', 4', 5', 6', cells of the various layers magnified 350 diameters. M'M' Medulla. In the medulla of Plate II. corpuscles of Deiter (cellules araignées, Gratiolet) are seen.





1



5



THE WEIGHT OF THE BRAIN

IN THE INSANE.

BY CROCHLEY CLAPHAM, L.R.C.P. LOND. &c.

WEST RIDING ASYLUM.

THE present Paper is in continuation of one published by me on the same subject in a former number of these Reports,¹ and deals with 484 brains, making, with the 716 already considered, a total of 1200 cases.

I propose to adopt the same method of discussing the subject as before employed, so as to facilitate reference. The only points of dissimilarity are occasioned by the introduction of a few more headings, and the substitution of grammes (French) for ounces, in the expression of the weights.

I shall give the weights, 1st, for the last 484 cases, and 2nd, for the whole 1200, in the following order. (1.) Sex; (2.) Age; (3.) Disease; (4.) Religious Persuasion.

1. SEX.

Number examined 1200. Male 701. Female 499. The average weight of the male brain was considerably higher than that of the female.

Average brain,² *male and female*, (*a*) = 1291·5 grms.

C. P. M. = 169·9 grms.; average age = 47·350 years.

¹ Vol. iii. 1873.

² Brain is used to signify the whole encephalon; C. P. M. to signify the cerebellum, pons, and medulla; (*a*) new cases; (*b*) total cases.

Ratio of C. P. M. to brain as 1 to 7.59.

(b.) Brain = 1303.835 grms.; C. P. M. = 169.7 grms.; average age = 46.786 years.

Ratio of C. P. M. to brain as 1 to 7.68.

Average brain, *male* (a) = 1310.228 grms.; C. P. M. = 176.5 grms.; average age = 46.496 years.

Ratio of C. P. M. to brain as 1 to 7.42.

(b.) Brain = 1356.061 grms.; C. P. M. = 176.9 grms.; average age = 46.419 years.

Ratio of C. P. M. to brain as 1 to 7.66.

Average brain, *female* (a) = 1208.278 grms.; C. P. M. = 159.3 grms.; average age = 48.718 years.

Ratio of C. P. M. to brain as 1 to 7.58.

(b.) Brain = 1230.466 grms.; C. P. M. = 159.4 grms.; average age = 47.301 years.

Ratio of C. P. M. to brain as 1 to 7.71.

The average difference between male and female brains was (a) 101.950 grms. (= 3.595 oz.); and (b) 125.595 grms. (= 4.430 oz.), in favour of the men.

Dr. Robert Boyd, in a recent paper¹ on Brain Weights, says that the insane male brain averages from 4 to 5 oz. heavier than that of the female, and this statement is borne out by my total averages.

It will also be seen from the above figures that the C. P. M. were not only actually, but, as compared with the encephalon, relatively, larger in men than in women.

The largest male brain in new cases occurred in a melancholic 45 years of age, and weighed 1729.319 grms. (= 61 oz.). The C. P. M. were not weighed as the brain was preserved entire. Another insane brain of the same weight is recorded in my last paper on this subject. The largest female insane brain (a) occurred in a case of senile dementia 85 years of age, and weighed 1555 grms. (= 54.851 oz.). This was an immense brain, and allowing a loss of 1 oz. for each decade after 40 years, its original weight must have been over 59½ ounces. (b) Largest female brain occurred in a case of mania 33 years of age = 1587 grms. (= 56 oz.), The

¹ 'British Medical Journal,' September 30, 1876.

smallest brain in the whole collection occurred in a female general paralytic 45 years of age, and weighed only 823 grms. ($= 29.030$ oz.), and the difference between it and the largest $= 906$ grms., or over 32 oz.

The largest male C. P. M. occurred in a case of general paralysis 51 years of age, and weighed 219.3 grms. ($= 7.735$ oz.), and bore a proportion to its encephalon of 1 to 6.133.

The largest female C. P. M. occurred in a case of organic dementia 44 years of age, and weighed 262.2 grms. ($= 9\frac{1}{4}$ oz.), bearing a ratio to its encephalon of 1 to 4.919, and being apparently normal in structure.

Smallest male C. P. M. $= 113.3$ grms.

Smallest female C. P. M. $= 85$ grms.

Extreme difference brain, *male* $= 761$ grms.

„ „ „ *female* $= 764$ „

„ „ C. P. M. *male* $= 106$ „

„ „ „ *female* $= 177.2$ „

Out of the 1200 brains examined, 4 weighed 1700 grms. ($= 60$ oz.) and upwards, and 45 of them weighed 1559 grms. ($= 55$ oz.) and upwards, 2 of the latter being female.

2. AGE.

I have considered the weight for age in decades of years, taking 0 and 9 as the initial and terminal figures in each instance up to 70 years. Cases over 70 are considered together.

UNDER 20 YEARS.

Number examined 36. Male 23. Female 13.

Average brain, *male and female* (*b*) $= 1264.8$ grms.; C. P. M. $= 166.4$ grms.; average age $= 17.1$ years.

Ratio of C. P. M. to brain as 1 to 7.60.

Average brain, *male* (*a*) $= 1341.4$ grms.; C. P. M. $= 167.7$ grms.; average age $= 16.7$ years.

(*b*.) Brain $= 1313.434$ grms.; C. P. M. $= 173.0$ grms.; average age $= 17.043$ years.

Ratio of C. P. M. to brain as 1 to 7.53.

Average brain, *female* (*a*) = 1103·25 grms.; C. P. M. = 160·6 grms.; average age = 15·25 years.

(*b.*) Brain = 1179 grms.; C. P. M. = 155·9 grms.; average age = 17·230 years.

Ratio of C. P. M. to brain as 1 to 7·62.

Largest brain = 1605 grms.; largest C. P. M. = 201 grms.

Smallest „ = 900 „ smallest „ = 127·5 „

Extreme difference, brain = 705 grms.; C. P. M. 73·5 grms.

BETWEEN 20 AND 30.

Number examined 106. Male 54. Female 52.

Average brain, *male and female* (*b*) = 1312·571 grms.; C. P. M. = 167·9 grms.; average age = 25·352 years.

Ratio of C. P. M. to brain as 1 to 7·81.

Average brain, *male* (*a*) = 1354·037 grms.; C. P. M. = 173 grms.; average age = 25·6 years.

(*b.*) Brain = 1365·018 grms.; C. P. M. = 173·0 grms.; average age = 25·685 years.

Ratio of C. P. M. brain as 1 to 7·88.

Average brain, *female* (*a*) = 1222 grms.; C. P. M. = 159·7 grms.; average age = 23·90 years.

(*b.*) Brain = 1257·039 grms.; C. P. M. = 162·2 grms.; average age = 25 years.

Ratio of C. P. M. to brain as 1 to 7·74.

Largest brain = 1658 grms.; largest C. P. M. = 212·6 grms.

Smallest „ = 900 „ smallest „ = 110·3 „

Extreme difference, brain 758 grms.; C. P. M. 102·3 grms.

BETWEEN 30 AND 40.

Number examined 265. Male 158. Female 107.

Average brain *male and female* (*b*) = 1301·011 grms.; C. P. M. = 172·0 grms.; average age = 35·052 years.

Ratio of C. P. M. to brain as 1 to 7·56.

Average brain, *male* (*a*) = 1314 grms; C. P. M. = 177·2 grms.; average age = 35·751 years

(*b.*) Brain 1341·132 grms.; C. P. M. = 180·1 grms.; average age = 35·474 years.

Ratio of C. P. M. to brain as 1 to 7.44.

Average brain, *female* (a) = 1219.261 grms.; C. P. M. = 161.3 grms.; average age = 34.214 years.

(b). Brain = 1241.766 grms.; C. P. M. = 160.3 grms.; average age = 34.429 years.

Ratio of C. P. M. to brain as 1 to 7.74.

Largest brain = 1644 grms.; largest C. P. M. = 226.7 grms.

Smallest brain = 923 grms.; smallest C. P. M. = 127.5 grms.

Extreme difference, brain, 721 grms.; C. P. M., 99.2 grms.

BETWEEN 40 AND 50.

Number examined 284. Male 180. Female 104.

Average brain, *male and female* (b) = 1315.426 grms.; C. P. M. = 172.8 grms.; average age = 44.355 years.

Ratio of C. P. M. to brain as 1 to 7.61.

Average brain, *male* (a) = 1346.042 grms.; C. P. M. = 179.9 grms.; average age = 44.014 years.

(b.) Brain = 1360.51 grms.; C. P. M. = 179.0 grms.; average age = 44.43 years.

Ratio of C. P. M. to brain as 1 to 7.60.

Average brain, *female* (a) = 1201.236 grms.; C. P. M. = 162.4 grms.; average age = 44.473 years.

(b). Brain = 1237.394 grms.; C. P. M. = 162.7 grms.; average age = 44.221 years.

Ratio of C. P. M. to brain as 1 to 7.60.

Largest brain = 1729 grms.; largest C. P. M. = 262.2 grms.

Smallest „ = 823 grms.; smallest „ = 113.3 „

Extreme difference: brain, 906 grms.; C. P. M., 148.9 grms.

In this decade occur the largest and smallest brains, and the largest C. P. M. of the series; also the greatest difference in C. P. M.

BETWEEN 50 AND 60.

Number examined 224. Male 128. Female 96.

Average brain, *male and female*, (b) = 1308.843 grms.; C. P. M. = 169.8 grms.; average age = 54.138 years.

Ratio of C. P. M. to brain as 1 to 7·70.

Average brain, *male* (a) = 1350·312 grms.; C. P. M. = 176·8 grms.; average age = 53·562 years.

(b). Brain = 1372·343 grms.; C. P. M. = 176·6 grms.; average age = 53·975 years.

Ratio of C. P. M. to brain as 1 to 7·77.

Average brain, *female* (a) = 1212·270 grms.; C. P. M. = 159·5 grms.; average age = 54·189 years.

(b). Brain = 1224·164 grms.; C. P. M. = 159·9 grms.; average age = 54·406 years.

Ratio of C. P. M. to brain as 1 to 7·65.

Largest brain = 1686 grms.; largest C. P. M. = 219·3 grms.

Smallest „ = 926 „ smallest „ = 113·3 „

Extreme difference: brain, 760 grms.; C. P. M. = 106 grms.

BETWEEN 60 AND 70.

Number examined 178. Male 108. Female 70.

Average brain, *male and female* (b) = 1303·359 grms.; C. P. M. = 167·8 grms.; average age = 64·370 years.

Ratio of C. P. M. to brain as 1 to 7·76.

Average brain, *male* (a) = 1354·411 grms.; C. P. M. = 175·1 grms.; average age = 64·529 years.

(b). Brain = 1352·431 grms.; C. P. M. = 174·8 grms.; average age = 64·157 years.

Ratio of C. P. M. to brain as 1 to 7·73.

Average brain, *female* (a) = 1211·5 grms.; C. P. M. = 153·3 grms.; average age = 64·692 years.

(b). Brain = 1227·571 grms.; C. P. M. = 155·8 grms.; average age = 64·7 years.

Ratio of C. P. M. to brain as 1 to 7·87.

Largest brain = 1644 grms.; largest C. P. M. = 226·7 grms.

Smallest „ = 850 „ smallest „ = 118·0 „

Extreme difference: brain, 794 grms.; C. P. M. 108·7 grms.

70 YEARS AND UPWARDS.

Number examined 85. Male 36. Female 49.

Average brain, *male and female* (b) = 1268·917 grms.; C. P. M. = 159·2 grms.; average age = 74·647 years.

Ratio of C. P. M. to brain as 1 to 7·97.

Average brain, *male* (*a*) = 1377·4 grms.; C. P. M. = 169·9 grms.; average age = 73·72 years.

(*b.*) Brain = 1352·94 grms.; C. P. M. = 169·8 grms.; average age = 74·583 years.

Ratio of C. P. M. to brain as 1 to 7·96.

Average brain, *female* (*a*) = 1215·863 grms.; C. P. M. = 155·8 grms.; average age = 74·954 years.

(*b.*) Brain = 1207·183 grms.; C. P. M. = 151·1 grms.; average age = 74·693 years.

Ratio of C. P. M. to brain as 1 to 7·98.

Largest brain = 1729 grms.; largest C. P. M. = 212·6 grms.

Smallest „ = 959 „, smallest „ = 85 „

Extreme difference: brain, 770 grms.; C. P. M., 127·6 grms.

This decade contains the largest sized brain (equalled by one in the decade of 40 to 50), and the smallest C. P. M. in the tables.

AGE UNKNOWN.

Number examined 22. Male 15. Female 7.

Average brain *male*, and *female* (*b*) = 1298·318 grms.; C. P. M. = 175·7 grms.

Ratio of C. P. M. to brain as 1 to 7·38.

Average brain, *male* (*a*) = 1331 grms.; C. P. M. = 173·9 grms.

(*b.*) Brain = 1362·93 grms.; C. P. M. = 176·8 grms.

Ratio of C. P. M. to brain as 1 to 7·70.

Average brain, *female* (*a*) = 1150·4 grms.; C. P. M. = 165·3 grms.

(*b.*) Brain = 1145·571 grms.; C. P. M. = 172·9 grms.

Ratio of C. P. M. to brain as 1 to 6·62

Largest brain = 1530 grms.; largest C. P. M. = 212·6 grms.

Smallest „ = 1035 „, smallest „ = 141·7 „

Extreme difference: brain, 495 grms.; C. P. M., 70·9 grms.

DISEASE.

Idiocy.

This division includes only Idiots pure and simple, Epileptic Idiots coming under the head of Epileptic Insanity.

Number examined 19. Male 11. Female 8.

Average brain, *male and female* (*a*) = 1148·3 grms.; C. P. M. = 153·16 grms.; average age = 23·875 years.

(*b.*) Brain = 1148·947 grms.; C. P. M. = 156·7 grms.; average age = 21·94 years.

Ratio of C. P. M. to brain as 1 to 7·33.

Average brain, *male* (*a*) = 1222 grms.; C. P. M. = 153·5 grms.; average age = 24·5 years.

(*b.*) Brain = 1200·72 grms.; C. P. M. = 162·4 grms.; average age = 22·81 years.

Average brain, *female* (*a*) = 1089·4 grms.; C. P. M. = 153 grms.; average age = 23·25 years.

(*b.*) Brain = 1077·75 grms.; C. P. M. = 149·23 grms.; average age = 20·571 years.

Largest brain = 1530 grms.; largest C. P. M. = 170 grms.

Smallest „ = 900 „ smallest „ = 141·7 grms.

Extreme difference: brain, 630 grms; C. P. M., 28·3 grms.

In the Pathological Museum of this Asylum is a brain of an Idiot weighing only 300 grms. (= 10·582 oz.). It was sent here from Inverness District Asylum.

The range of difference in the C. P. M. is least in this disease, and with the exception of General Paralysis it is the disease showing the greatest average proportion of C. P. M. to brain.

Imbecility (Simple).

Number examined 11. Male 5. Female 6.

Average brain, *male and female* (*a*) = 1236·3 grms.; C. P. M. = 170·9 grms.; average age = 40·6 years.

(*b.*) Brain = 1285·09 grms.; C. P. M. = 174·6 grms.; average age = 36·2 years.

Ratio of C. P. M. to brain as 1 to 7·36.

Average brain, *male* (*a*) = 1255 grms.; C. P. M. = 172·1 grms.; average age = 37 years.

(b.) Brain = 1331·2 grms.; C. P. M. = 179·8 grms.; average age = 36 years.

Average brain, *female* (a) = 1217·6 grms.; C. P. M. = 167·3 grms.; average age = 43 years.

(b.) Brain = 1246·6 grms.; C. P. M. = 161·6 grms.; average age = 36·3.

Largest brain = 1474 grms.; largest C. P. M. = 198·4 grms.

Smallest „ = 997 „ smallest „ = 155·9 „

Extreme difference: brain, 477 grms.; C. P. M., 42·5 grms.

Some remarkably large brains have been found amongst Imbeciles. One in particular, weighed by Dr. Levinge when at the Hants County Asylum, and the notes of which he purposes publishing at an early date, was $70\frac{1}{2}$ oz. (= 1998 grms.) in weight. This is, I believe, the heaviest brain on record. It occurred in a patient 30 years of age, a congenital imbecile, and was unfortunately sliced before being weighed. Its consistence was normal, and there was no evidence of sclerosis. The Cerebrum alone weighed 1786 grms. (= 63 oz.) and the C. P. M. alone 212·6 grms. (= $7\frac{1}{2}$ oz.). Ratio of C. P. M. to Encephalon as 1 to 9·39. The patient, who was 5 ft. 3 in. in height, died of Phthisis. Dimensions of head were as follows: Whole circumference, $24\frac{1}{2}$ in.; Arch, ant.-post., $15\frac{1}{2}$ in.; Transverse, $12\frac{1}{2}$ in.

Dementia (Simple).

Number examined 254. Male 159. Female 95.

Average brain, *male and female* (a) = 1288·058 grms.; C. P. M. = 167·9 grms.; average age = 48·204 years.

(b.) Brain = 1310·956 grms.; C. P. M. = 169·7 grms.; average age = 49·132 years.

Ratio of C. P. M. to brain as 1 to 7·72.

Average brain, *male* (a) = 1339·419 grms.; C. P. M. = 173·7 grms.; average age = 47·2 years.

(b.) Brain = 1356·874 grms.; C. P. M. = 175·9 grms.; average age = 49·429 years.

Average brain, *female* (a) = 1208·45 grms.; C. P. M. = 158·9 grms.; average age = 49·789 years.

(b.) Brain = 1234·105 grms.; C. P. M. = 153·7 grms.; average age = 48·638 years.

Largest brain = 1715 grms.; largest C. P. M. = 226·7 grms.

Smallest „ = 878 „ smallest „ = 113·3 „

Extreme difference: brain, 837 grms.; C. P. M., 113·4 grms.

Dementia (Senile).

In my former paper both Senile and Organic Dementia were included under this head, whereas I have now distinguished them as regards the last 484 cases. I shall first give them altogether as before, and then the organic dementia separately.

Number examined 209. Male 107. Female 102.

Average brain, *male and female* (a) = 1287·146 grms.; C. P. M. = 167·1 grms.; average age = 62·055 years.

(b.) Brain = 1278·382 grms.; C. P. M. = 163·8 grms.; average age = 64·843 years.

Ratio of C. P. M. to brain as 1 to 7·80.

Average brain, *male* (a) = 1349·540 grms.; C. P. M. = 174·2 grms.; average age = 60·364 years.

(b.) Brain = 1348·504 grms.; C. P. M. = 172·9 grms.; average age = 63·140 years.

Ratio of C. P. M. to brain as 1 to 7·79.

Average brain, *female* (a) = 1204·696 grms.; C. P. M. = 157·5 grms.; average age = 64·415 years.

(b.) Brain = 1204·901 grms.; C. P. M. = 153·8 grms.; average age = 66·704 years.

Ratio of C. P. M. to brain as 1 to 7·83.

Largest brain = 1729 grms.; largest C. P. M. = 212·6 grms.

Smallest „ = 850 „ smallest „ = 85 „

Extreme difference: brain, 879 grms.; C. P. M., 127·6 grms.

Dementia (Organic).

Number examined 59. Male 37. Female 22.

Average brain, *male and female* (a) = 1291·949 grms.; C. P. M. = 170·5 grms.; average age = 53·810 years.

Ratio of C. P. M. to brain as 1 to 7·57.

Average brain, *male* (*a*) = 1347·783 grms.; C. P. M. = 176·9 grms.; average age = 53·027 years.

Average brain, *female* (*a*) = 1198·045 grms.; C. P. M. = 158·4 grms.; average age = 55·190 years.

Mania, Melancholia, and Acute forms of Insanity.

Number examined 235. Male 104. Female 131.

Average brain, *male and female* (*a*) = 1338·15 grms.; C. P. M. = 172·8 grms.; average age = 43·338 years.

(*b.*) Brain = 1350·425 grms; C. P. M. = 172·8 grms.; average age = 42·082 years.

Ratio of C. P. M. to brain as 1 to 7·81.

Average brain, *male* (*a*) = 1431·705 grms.; C. P. M. = 183·2 grms.; average age = 48·225 years.

(*b.*) Brain = 1441·615 grms.; C. P. M. = 183·3 grms.; average age = 45·11 years.

Average brain, *female* (*a*) = 1244·588 grms.; C. P. M. = 163·0 grms.; average age = 38·880 years.

(*b.*) Brain = 1278·030 grms.; C. P. M. = 164·1 grms.; average age = 39·753 years.

Largest brain = 1729 grms.; largest C. P. M. = 226·7 grms.

Smallest „ = 865 „, smallest „ = 113·3 „,

Extreme difference: brain, 864 grms.; C. P. M., 113·4 grms.

General Paralysis.

Number examined 243. Male 197. Female 46.

Average brain, *male and female* (*a*) = 1264·53 grms.; C. P. M. = 175·5 grms.; average age = 41·365 years.

(*b.*) Brain = 1270·271 grms.; C. P. M. = 174·0 grms.; average age = 41·610 years.

Ratio of C. P. M. to brain as 1 to 7·30.

Average brain, *male* (*a*) = 1279·3 grms.; C. P. M. = 178·6 grms.; average age = 41·285 years.

(*b.*) Brain = 1302·015 grms.; C. P. M. = 177·5 grms.; average age = 41·768 years.

Average brain, *female* (*a*) = 1110·095 grms.; C. P. M. = 163·1 grms.; average age = 42·2 years.

(*b.*) Brain = 1134·304 grms.; C. P. M. = 159·3 grms.; average age = 40·93 years.

Largest brain = 1667 grms.; largest C. P. M. = 255.1 grms.

Smallest „ = 823 „ smallest „ = 127.5 „

Extreme difference: brain 844 grms.; C. P. M. 127.6 grms.

The smallest brain in the series occurred in this disease.

Epileptic Insanity.

Number examined 117. Male 65. Female 52.

Average brain, *male and female* (*a*) = 1231.865 grms.;
C. P. M. = 163.0 grms.; average age = 32.461 years.

(*b.*) Brain = 1314.410 grms.; C. P. M. = 164.4 grms.;
average age = 36.646 years.

Ratio of C. P. M. to brain as 1 to 7.99.

Average brain, *male* (*a*) = 1388.117 grms.; C. P. M. =
170.2 grms.; average age = 29.676 years.

(*b.*) Brain = 1391.646 grms.; C. P. M. = 173.3 grms.
average age = 31.171 years.

Average brain, *female* (*a*) = 1214.5 grms.; C. P. M. =
150.0 grms.; average age = 37.72 years.

(*b.*) Brain = 1217.673 grms.; C. P. M. = 154.3 grms.;
average age = 36.692 years.

Largest brain = 1630 grms.; largest C. P. M. = 226.7 grms.

Smallest „ = 850 grms.; smallest; „ = 110.3 grms.

Extreme difference: brain, 780 grms.; C. P. M., 116.4 grms.

Chronic Mania.

This includes all cases of Chronic delusional insanity.

Number examined 112. Male 53. Female 59.

Average brain, *male and female* (*a*) = 1325.174 grms.;
C. P. M. = 171.9 grms.; average age = 45.126 years.

(*b.*) Brain = 1327.267 grms.; C. P. M. = 171.9 grms.;
average age = 46.863 years.

Ratio of C. P. M. to brain as 1 to 7.72.

Average brain, *male* (*a*) = 1386.636 grms.; C. P. M.
= 179.9 grms.; average age = 47.09 years.

(*b.*) Brain = 1392.924 grms.; C. P. M. = 179.2 grms.;
average age = 47.431 years.

Average brain, *female* (*a*) = 1257.56 grms.; C. P. M. =
162.7 grms.; average age = 43.03 years.

(b.) Brain = 1268·288 grms.; C. P. M. = 164·8 grms.; average age = 46·372 years.

Largest brain = 1697 grms.; largest C. P. M. = 212·6 grms.

Smallest „ = 990 grms.; smallest „ = 127·6 grms.

Extreme difference : brain, 707 grms.; C. P. M., 85 grms.

Brain Wasting.

In 59 cases, brain wasting was a marked feature on *post mortem* examination. Of these 29 were male and 30 female.

Average brain, *male and female* = 1256·644 grms.; C. P. M. = 164·3 grms.; average age = 60·929 years.

Ratio of C. P. M. to brain as 1 to 7·64.

Average brain, *male* = 1251·241 grms.; C. P. M. = 172·3 grms.; average age = 57·392 years.

Ratio of C. P. M. to brain as 1 to 7·26.

Average brain, *female* = 1195·2 grms.; C. P. M. = 156·6 grms.; average age = 64·337 years.

Ratio of C. P. M. to brain as 1 to 7·63.

If the foregoing figures be compared with the averages for the corresponding ages in the Age Section, it will be seen that, in the case of *males*, though the brain has dropped 121·102 grammes, or 8·824 per cent. below the average weight, the C. P. M. have only fallen 4·3 grammes, or 2·434 per cent., whilst in the case of *females*, the C. P. M. stand $\frac{4}{5}$ th of a gramme above the corresponding average, the brain being 32·371 grammes below it—a loss of 2·636 per cent. From the above it would appear that the Cerebellum, Pons, and Medulla are comparatively little affected, as regards weight, by brain wasting—a fact already pointed out by Dr. Robert Boyd in his paper cited above.

RELIGIOUS PERSUASION.

The last 500 cases (304 males and 196 females) I have tabulated according to their religious persuasion. I have classed them as follows :

- (1) Church of England.
- (2) Protestant Dissent.
- (3) Roman Catholic.

(4) No Religion.

(5) Religion Unknown.

Church of England.

212 examples, 124 men and 88 women.

Average brain, *male and female* = 1228·188 grms.;

C. P. M. = 170·0 grms.; average age = 46·319 years.

Ratio of C. P. M. to brain as 1 to 7·22.

Average brain, *male* = 1328·685 grms.; C. P. M. = 176·6 grms.; average age = 44·601 years.

Ratio of C. P. M. to brain as 1 to 7·52.

Average brain, *female* = 1200·215 grms.; C. P. M. = 160·0 grms.; average age = 48·712 years.

Ratio of C. P. M. to brain as 1 to 7·50.

Protestant Dissent.

197 examples, 127 men and 70 women.

Average brain, *male and female* = 1298·416 grms.;

C. P. M. = 169·1 grms.; average age = 49·751 years.

Ratio of C. P. M. to brain as 1 to 7·67.

Average brain, *male* = 1344·102 grms.; C. P. M. = 174·4 grms.; average age = 49·531 years.

Ratio of C. P. M. to brain as 1 to 7·70.

Average brain, *female* = 1215·528 grms.; C. P. M. = 160·2 grms.; average age = 50·164 years.

Ratio of C. P. M. to brain as 1 to 7·58.

Roman Catholic.

40 examples, 21 men and 19 women.

Average brain, *male and female* = 1304·625 grms.; C. P. M. = 170·1 grms.; average age = 43 years.

Ratio of C. P. M. to brain as 1 to 7·66.

Average brain, *male* = 1370·142 grms.; C. P. M. = 183·3 grms.; average age = 41·3 years.

Ratio of C. P. M. to brain as 1 to 7·47.

Average brain, *female* = 1232·210 grms.; C. P. M. = 153·6 grms.; average age = 44·94 years.

Ratio of C. P. M. to brain as 1 to 8·02.

No Religion.

27 examples, 17 men and 10 women.

Average brain, *male and female* = 1255·814 grms.; C. P. M. = 166·0 grms.; average age = 41·4 years.

Ratio of C. P. M. to brain as 1 to 7·56.

Average brain, *male* = 1357·235 grms.; C. P. M. = 179·9 grms.; average age = 42·237 years.

Ratio of C. P. M. to brain as 1 to 7·54.

Average brain, *female* = 1083·4 grms.; C. P. M. = 140·1 grms.; average age = 39·8 years.

Ratio of C. P. M. to brain as 1 to 7·73.

Here the brain average is lowered by the number of Idiots included.

Religion Unknown.

24 examples, 15 men and 9 women.

Average brain, *male and female* = 1350·291 grms.; C. P. M. = 167·9 grms.; average age = 52·428 years.

Ratio of C. P. M. to brain as 1 to 8·04.

Average brain, *male* = 1423·06 grms.; C. P. M. = 174·5 grms.; average age = 53·461 years.

Ratio of C. P. M. to brain as 1 to 8·15.

Average brain, *female* = 1229 grms.; C. P. M. = 156·8 grms.; average age = 50·75 years.

Ratio of C. P. M. to brain as 1 to 7·83.

In the three great divisions of Christianity as professed in this country—Church of England, Protestant Dissent, and Roman Catholicism—it is curious to find that not only have the Roman Catholics heavier brains than the Protestant Dissenters, and these again than those of the Church party, but also that the Cerebellum, Pons, and Medulla, as compared with the entire brain, are proportionately larger in the Church of England cases than in the Roman Catholics. And this is true of the women as well as of the men, as will be seen on reference to the foregoing figures. Of course my experience is only of the brains of insane members of these classes, but judging from the general correspondence, as regards weight, between sane and insane brains, I see no reason to doubt that the same relation will hold good in the case of those enjoying their liberty.

Average Weight of Encephalon, and of Cerebellum, Pons, and Medulla, in 1,200 Cases of Insanity:—

	ALI—MALE AND FEMALE				MALE			FEMALE			Ratio of C. P. M. to Encephalon (All)
	Encephalon	C. P. M.	Age	grammes	Encephalon	C. P. M.	Age	Encephalon	C. P. M.	Age	
<i>Age.</i>											
Under 20 years	1264.8	166.4	17.1	1313.434	173.0	17.043	1179.000	155.9	17.230	1. to 7.60	
20 to 30 "	1312.571	167.9	25.352	1365.018	173.0	25.685	1257.039	162.2	25.000	1. to 7.81	
30 to 40 "	1301.011	172.0	35.052	1341.132	180.1	35.474	1241.766	160.3	34.429	1. to 7.56	
40 to 50 "	1315.426	172.8	44.355	1360.51	179.0	44.43	1237.394	162.7	44.221	1. to 7.61	
50 to 60 "	1308.843	169.8	54.138	1372.343	176.6	53.975	1224.164	159.9	54.406	1. to 7.70	
60 to 70 "	1303.359	167.8	64.370	1352.481	174.8	64.157	1227.571	155.8	64.7	1. to 7.76	
70 years and upwards	1268.917	159.2	74.647	1352.94	169.8	74.583	1207.183	151.1	74.693	1. to 7.97	
Age unknown	1298.318	175.7	...	1362.93	176.8	...	1145.571	172.9	...	1. to 7.38	
All ages .	1303.835	169.7	46.786	1356.061	176.9	46.419	1230.466	159.4	47.301	1. to 7.68	
<i>Disease.</i>											
Idiocy .	1148.947	156.7	21.94	1200.72	162.4	22.81	1077.75	149.2	20.571	1. to 7.33	
Imbecility .	1285.09	174.6	36.2	1331.2	179.8	36.000	1246.6	161.6	36.3	1. to 7.36	
Dementia (simple) .	1310.956	169.7	49.132	1356.874	175.9	49.429	1234.105	158.7	48.638	1. to 7.72	
" (senile) .	1278.382	163.8	64.843	1348.504	172.9	63.140	1204.901	153.8	66.704	1. to 7.80	
" (organic) .	1291.949	170.5	53.810	1347.783	176.9	53.027	1198.045	158.4	55.190	1. to 7.57	
Mania, Melancholia, and acute forms	1350.425	172.8	42.082	1441.615	183.3	45.11	1278.030	164.1	39.753	1. to 7.81	
General Paralysis	1270.271	174.0	41.610	1302.015	177.5	41.768	1134.304	159.3	40.93	1. to 7.30	
Epileptic Insanity	1314.410	164.4	36.646	1391.646	173.3	31.171	1217.673	154.3	36.692	1. to 7.99	
Chronic Mania	1327.267	171.9	46.863	1392.924	179.2	47.431	1268.288	164.8	46.372	1. to 7.72	
Brain Wasting	1256.644	164.3	60.929	1251.241	172.3	57.392	1195.2	156.6	64.337	1. to 7.64	
<i>Religion.</i>											
Church of England	1228.188	170.0	46.319	1328.685	176.6	44.601	1200.215	160.0	48.712	1. to 7.22	
Protestant Dissent .	1298.416	169.1	49.751	1344.102	174.4	49.531	1215.528	160.2	50.164	1. to 7.67	
Roman Catholic	1304.625	170.1	43.000	1370.142	183.3	41.3	1232.210	153.6	44.94	1. to 7.66	
No Religion .	1255.814	166.0	41.4	1357.235	179.9	42.237	1083.4	140.1	39.8	1. to 7.56	
Religion unknown .	1350.291	167.9	52.428	1423.06	174.5	53.461	1229.000	156.8	50.75	1. to 8.04	

To reduce to ounces divide by 28.3495.

ON
CLASSIFICATION AND NOMENCLATURE
IN
NERVOUS DISORDERS.

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IN the following Paper I think it will be better to omit most of the references to authorities which it might be desirable to introduce, if this were the introduction to a classification of diseases in general, which it appears to me ought soon to be attempted, if Medicine is to assert and to attempt to establish her claim to be considered a science. After a good deal of consideration I have come to the conclusion that such a classification as will prove of permanent value must be etiological; and, although I am aware that high authority may be cited against this method, I shall not here do more than suggest the general considerations which seem to require that a truly scientific classification must be founded on Causes. Firstly, the problem of science in general is the investigation of Causes. The attempt to include a large number of phenomena under a general expression, or to discover the laws of phenomena, is only another way of making this statement; and this is the universally admitted scope and aim of science. Secondly, a large number of conditions known under the name of disease are indistinguishable from one another if their causes are not known. Yet their effects and causes are entirely different. For instance,

the group of diseases known as *Pyrexia* include the widely differing disorders known as Fevers, as Inflammations, and as Specific Inflammations. Unless the observer has time given him, he cannot by inspection differentiate these conditions from one another, but if he knows the causes then he can do so. The answer to the question whether a patient is going to have Typhus Fever or Pneumonia can be given if we know the antecedents as to exposure to cold and wet on the one hand, or to organic matter in a state of change on the other, but it cannot be given, except by waiting, if we don't know the relation with these conditions. Nothing in the symptomatology will differentiate the one from the other in the earliest stage. An incidental indication of the value of the etiological over other methods of classification is shown in the repeated occurrence of the following interesting quotation from the Official Nomenclature of Diseases, drawn up by a Joint Committee appointed by the Royal College of Physicians of London, and adopted by the Registrar-General. The system of classification there adopted is 'that based upon anatomical considerations.' The following is the quotation, repeated on twenty-eight different pages of the classification, and referring to about fifty different diseases:—'When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.' From this it appears that a classification 'based upon anatomical considerations' is compelled in numerous cases to refer back to causes.

The division of Causes into Exciting and Predisposing is no doubt sound, and must be founded on; and speaking generally, it seems to me that in Acute Diseases, or what are called so (for I shall immediately offer reasons for analysing the notions Acute and Chronic into what seem to me to be their two very different factors), the exciting causes are predominant, and almost entirely determine the nature of the affections, while in Chronic Diseases (so-called), the exciting causes count for comparatively very little, and the nature of these diseases is determined chiefly by the constitutional and hereditary causes—the predisposing of older writers.

Three requisites seem to be demanded in a sound classification. Firstly, it must be exhaustive and not tautological. Secondly, it must be on a thoroughgoing plan, or, as the logicians express it, it must not contain cross-divisions. Thirdly, it must group together diseases whose characters are alike. In short, the classification must be natural. One or more of these principles appears to me to have been violated in all the attempts at classification which I have examined. And in addition many of them seem to fall into the error of mistaking steps in the course of disease for new genera. To name Catarrhal Ophthalmia and Purulent Ophthalmia, and to differentiate both from Conjunctivitis, seems to me to be analogous to distinguishing a flowering *Iroliius* from an ached *Ranunculus* in Botany, and to differentiating both from a *Potentilla*. Properly speaking, such an attempt at classification as is here spoken of ought to be preceded by a chapter on Terms, since these are used very loosely in current medical literature both by different authors and by the same authors at different times; but in order that this Paper may not extend to such a length as would render it inadmissible for its present purpose, only such considerations regarding the meaning of terms will be here introduced as seem absolutely necessary. I shall here, therefore, only deal with the terms *Acute* and *Chronic* as applied to disorders, and with the various notions comprehended under the term *Constitutional*, before proceeding to say what are the great facts in nervous disorders on which, in my opinion, our classification ought to be based. *Acute* and *Chronic*, we are told in all the histories of medicine, were introduced by Asclepiades of Bithynia, yet the adjective $\acute{o}\xi\upsilon\varsigma$ is used by Hippocrates to denote disorders which are severe. A secondary meaning of $\acute{o}\xi\upsilon\varsigma$ is *quick*, or *rapid*, and hence, as Sydenham points out, an *acute* disease (*acute* being the Latin equivalent of $\acute{o}\xi\upsilon\varsigma$) is one which is severe and runs its course rapidly. On the other hand, a *chronic* disorder is milder and drags a slow length along. Here then, it is evident to anyone who thinks upon the matter, we have two very different notions covered by a single term, viz. severity and shortness, as opposed to mildness and length. It would

seem to be desirable to separate these two ideas, which are not *ideas* only, since they refer to *facts* in the course of diseases; and what I would propose is the following. Let us retain the term *Acute* for disorders which run a severe course. Its opposite would then be *Mild*, a term which is already in use in medicine; while by the term *Sub-acute* we might indicate an intermediate group, whose existence is also recognised by the profession. The feature which appears to me most worthy of study in such diseases is the temperature; and I think so because heat production is the connecting link by which a larger and larger number of vital phenomena are being brought under the domain of the law of the Conservation of Energy, which is almost certain before long to vindicate its sway over all phenomena whatever. I propose, therefore, to define *Acute* disorders as all those in which the temperature reaches any point over 102·5° F. *Sub-acute* I propose should cover conditions in which the temperature is above 100° but not above 102·5° F.; while *Mild* diseases would be all those in which the temperature, though rising above normal, does not rise higher than 100° F. These seem to be the conditions to which these terms are at present generally applied, and they are conditions whose existence at least is matter of observation. These are, further, *all* the diseases of elevated temperature; and they are naturally opposed to diseases of diminished temperature, a set of phenomena which appear to me not to have obtained the study they deserve, although numerous incidental references to them exist in current literature. Next, as to the term *Chronic*. Properly speaking, this means long continued. The better Greek form would, no doubt, be represented by the English equivalent *Polychronic*, but it is well not to interfere with language more than is absolutely necessary, and I introduce the word here only that I may draw a distinction between it and its opposite in Platonic Greek, which may at once be rendered into English as *Brachychronic*, or of *short duration*.¹ Letting *Chronic* then stand as it is, we have its natural opposite in *Brachychronic*. It will be well to give to these terms as much definiteness as possible,

¹ In the *Timaeus*, 75 B, occurs the following passage:— . . . λογισζομένοις, πότερον πολυχρονιώτερον χείρον ἢ βραχυχρονιώτερον βέλτιον ἀπεργάσαιντο γένος.

and I therefore propose to define a brachychronic disease as one which lasts not longer than twenty-eight days, which is the natural length of Typhoid Fever, the longest of the diseases at present called *Acute*. A disease lasting longer than twenty-eight days would, on this definition, be chronic.¹ This seems to me also to be in keeping with observed facts. When Typhoid Fever, for example, has a course extending over forty days, as happens from time to time, it usually takes what in common language is called the chronic form. On this view it would be chronic in duration, while, as regards severity, it might be *acute*, *sub-acute*, or *mild*. It generally happens, though not always, that the latter portion of such a disease is sub-acute or even mild in intensity. Hence, no doubt, the reason that the division into *Acute* and *Chronic* has held its own for so long a time in medicine, although both terms cover conditions which are entirely dissimilar from one another, but whose dissimilarity is overlooked because of a tolerably constant connection in fact.² In passing, I may observe that it seems to me that the form taken by a disorder in a given instance, as regards both the length of its duration and its intensity, is determined mainly by the constitution, diathesis, and heredity of the given person, in the sense in which these terms will be immediately defined. Speaking generally, an acute disorder of brachychronic duration is the natural form of disease in persons who, and whose ancestors, have lived much in the open country air and under favourable conditions; while sub-acute and chronic disorders more naturally attack those persons who, and whose ancestors, have been subjected to town life and indoor occupations. And this leads to a statement of the definitions which it is proposed to give to the terms Constitution, Diathesis, and Heredity, and to which it seems desirable to restrict them for the future. In the life of a grown organism

¹ The reason for taking twenty-eight days as the duration of typhoid fever, not twenty-one as is commonly done, is because in fact the temperature in typhoid has not as a rule become normal before that time. In the ordinary account no notice is taken of the period of sub-normal temperature, a most important one nevertheless.

² I exclude from consideration the meaning of *recurrent*, which the term *Chronic* is sometimes made to cover. Such a use of the term is manifestly erroneous.

there are two main sets of facts to be considered. First, counting from the present time backwards, there are all the facts which have taken place in the history of the organism between the present time and birth. To this set of facts or inter-relations between the organism and external nature it is proposed to confine the term *Constitution*. The next great set of facts affecting the life of the individual is the family history. That, again, seems to break up naturally into the two groups of facts included in the intra-uterine life, and the facts of the previous family history proper. To the facts of the intra-uterine life it is proposed to confine the term *diathesis*, while *heredity*, it is suggested, should be employed to cover the facts of the family history. This restriction of the meanings of these terms, it is contended, would have several advantages over the present use of the words. Because, not to say that we should know exactly what we were talking about, the distinction founds on real facts in nature, and these facts, no doubt, do determine the form that (a) the healthy life, and (b) the disease phenomena, assume in given cases. In our classification it will be found that *varieties* in the form assumed by diseases are most frequently determined by the *diathesis* and the *heredity*. The *Constitution*, on the other hand, frequently determines the *Species*. What determine the *Genera*¹ and *Classes* will appear further on in the statement of the classification itself. Another advantage which the suggested limitations of meaning would have, would be that of suggesting to the careful physician limits of enquiry which he ought to take, and the answers to which would often determine his treatment. Lastly, such restrictions of meaning would be valuable as showing how it is that a Constitution is in constant process of change, while a Diathesis and Heredity are more or less fixed and determinate quantities, which no doubt have their influence on future constitutions, but are invariable for the one in question. In this manner the fundamental unity of successive organisations through family history obtains a prominence

¹ It would be very interesting, and would probably prove of real value, to discuss Cullen's opinion, that nature has formed only *species* and not *genera*, but it is impossible here. Was he correct?

which it would have been better for all of us to have more clearly perceived. In this connection, it will probably prove desirable to rearrange a set of terms which might be applied to constitutions, &c., founding on facts in nature; while if such terms were carefully defined, and their meanings restricted, real advantage would accrue to the study and practice of physic; but our present limits do not allow of this.

Before leaving the term Constitution it should be said that the word, as well as the adjectival form Constitutional, is often used in current literature as synonymous with General. In this case it is opposed to Local. For instance, we say that in certain local disorders, the constitution sympathises, meaning that the whole organism is affected. And we say that such an affection as syphilis is constitutional when we mean that it is general. It would be well to express such meanings by the term *general* so as to avoid confusion.

In the Nosology of Cullen occurs the following interesting passage:—‘In my opinion, the generality of morbid affections so depend on those of the nervous system, that almost every disease might be called nervous. A very convenient distinction, however, may be found; and those diseases only I call nervous which affect the nervous system alone, or at least in a primary way, without at the same time affecting either the circulation of the blood, or the nature of the humours, except in a secondary manner.’ As we should expect from such a statement, such diseases as *cerebritis*—inflammations of nervous substances—do not find a place in Cullen’s nosology of the nervous affections. The equivalent *phrenitis* he classes among the *Phlegmasiæ*, order *Phlogosis*. If the term *neuralgia* had been in use in his day, I suppose he would have classed the disorder referred to by that name under Class IV. *Locales*, order *Dysæsthesiæ*. But while the second part of Cullen’s statement seems open to manifest criticism, the former one only reflects greater glory on his great name, the further advance of time and knowledge having only further vindicated the paramount importance of the nervous system as the ruling system in all

the higher organisms. It seems necessary to make a preliminary observation, before proceeding to the classification itself. So far as possible, in nomenclature, only objective physical changes should be named, and subjectivities or even functional conditions as little as possible. The reason of this is obvious, being in short this: disorder of function of various portions of the nervous system may proceed from very various objective states, either of other portions of the nervous system, or of parts of the body in general. Disorder of the function of a given nerve or portion of nervous system does not necessarily imply objective physical change in that nerve or part of nervous system, and the functional name may or may not, therefore, afford us any information. Such a name as *tinnitus* for example, when given as a name to a disorder, is obviously an objectionable one, since in fact we know that *tinnitus* may be symptomatic of simple accumulation of wax in the outer ear; of blocking of the Eustachian tube; of disease of the parts of the middle ear; of affection of the internal ear or of the auditory nerve itself, or even of nerve centres from which it arises, or of other centres in communication with the last. The same kind of illustration might be drawn from the use of such a term as *amaurosis* or *amblyopia*, or from the use by older medical writers of terms like *coma*, *carus*, &c., who used to first create a mental conception of what *coma* or *carus* was, and then indulge in fine hair-splitting as to whether a given condition approximated or not to the metaphysical entity. Names given to disorder of function ought, indeed, always to be received with suspicion, and should at once raise the question whether the nature of the disease is known. As medicine progresses, it is certain that the tendency is to bring into greater prominence the objective physical changes and to sink always more and more the functional changes. This position would not in general be controverted, but in dealing with affections of the nervous system we encounter two special difficulties. The first is that functional disorder is generally more important in the case of the nervous system than in any other instance: that in fact it often seems to be the disease. The second is that we don't know in many cases what the physical changes

associated with functional disorders are. The conditions, for instance, called mania and paralysis illustrate both these propositions. They are both functional names, but they must both stand probably for a very long time. Mania is, strictly, excitement of intellectual centres. Coma, or dementia, its opposite, being complete absence of function of the same centres. But the excitement cannot be studied apart from its causes, and there are some forms of intellectual excitement that would hardly justify the term mania being applied to them. A certain kind of mania, for instance, is properly called alcoholism. Then again, we do not know what the precise centres affected in mania are, nor the mode in which they are affected. In the case of paralysis, say of sensation, to choose an instance which raises all the difficulties at once, the loss of the function of sensibility seems to be at once the disease and its functional manifestation; and our difficulty in distinguishing these two things is like the difficulty experienced by the old metaphysicians in differentiating the objective and subjective element in the secondary qualities of matter. In fact, with our present knowledge, the problem seems to be an insoluble one: but the general indication rises quite clearly before the enquirer, that he ought to seek for objective physical changes to which to affix his names, and that functional changes should not demand names, except temporarily and until the objective changes associated have been determined. If it were possible, for instance, to predicate hyperæmia or spanæmia of certain nerve centres, that would be a step in the scientific direction. The next question would be whether the hyperæmia or spanæmia was primary or secondary, and what was its cause.

When one carefully investigates the laws of organic action, one general law seems to assert itself as supreme, and that seems to be this.¹ All agents whatever capable of acting on the economy exert upon it at least a twofold and contrary action in time. If these actions be called primary and secondary, this law might be otherwise stated in this

¹ This law seems to hold even in organisms without a nervous system; but in the text its truth is assumed only for nervous organisations.

form:—The secondary effect of all agents capable of affecting the economy is contrary to the former. Illustrations of this law will be found in the tonico-relaxant effects of the application of cold and of many medicines; in the paralyso-stimulant effects of stimulants and narcotics; in the purgativo-constipating effects of purgative medicines; and in the spanæmico-congestive effects of such organic poisons as fever contagions. A curious implicate of it seems to be the statement that tonics are really depressants and that stimulants are really paralyzers. Conversely, in attempting to classify diseases of the nervous system, even after we have been able to see congestion or spanæmia, we ought to proceed to the further enquiry whether this condition was primary or secondary before we can be said to understand the condition. In fact, congestion is followed by spanæmia, and in practice it often happens that the medical man is consulted not when the patient's organs are in the original congested condition, but when a spanæmic condition has supervened. A name, such for instance as neuralgia, is then given to the patient's disorder, and the term is in turn apt to tyrannise over both the medical man and the patient. If neuralgia is a name proper to be given to the condition, such a state should be called spanæmic neuralgia, and differentiated from hyperæmic neuralgia. In many instances, however, even the name neuralgia seems an improper one, as improper or even more so than naming the dropsy and overlooking the heart or kidney affection which causes it, or as supposing that a limb amputated some years ago is the seat of sensations which seem to be in its extremities. An example will suffice to illustrate what is here meant. A medical man is consulted by a patient who complains of pain down the back of the thigh, and other symptoms which are generally termed sciatica. On enquiry he finds that sometimes the sciatica is replaced by lumbago, again by gastric neuralgia, again by facial; and that all or any of these may suddenly disappear under strong mental excitement. Further enquiry elicits the facts that the patient has for years been harassed by business, that he has been anxious and sleepless, and has eaten his food too

quickly, and worked too soon after having it. In short, a general nervous hyperæmia has existed for several years, and then the condition has given way to the present neuralgic one. With such a history, and the case is not by any means an uncommon one, it is more than doubtful whether we should call the condition one of neuralgia or nerve-chord affection at all. The real state seems to be central, and to be anæmia of the whole cerebro-spinal nervous centres. Phenomenally, the pain is in the nerve. Really, it is an affection of the centre, which, in accordance with all known facts in nervous organisation, is referred to the extremities of the nerves. And yet physicians of eminence are often content to call such an affection neuralgia, in place of making out what it really is, namely, nervous spanæmia following nervous hyperæmia. Considerations of this sort show how exceedingly unscientific such terms as hyperæsthesia are, since excess of sensibility may be due either to hyperæmia or spanæmia of the hyperæsthetic part, and the name gives no indication of the real nature of the affection. Of course names come to lose their primary significations, and to stand as mere symbols in time. Hence they do not do so much harm as might be anticipated. Enough, however, has, it is hoped, been said to justify suspicion of all merely functional names, and to show their temporary character if they are admissible at all. The quiet dropping out of medical literature of such a disease as used to be described under the name of Nostalgia must be looked upon as a healthy sign, but the principles which condemn that name might with advantage eliminate a number of others if they were rigorously applied. In short, classification on the basis of such characters is as much to be condemned as a similar attempt in botany, based upon such a fleeting, changeable, and evanescent character as the colours of the flower.

To apply these general considerations to the classification of disorders of the nervous system, the following general statements seem to be sufficient. Disease is one sub-kingdom in the kingdom of Conditions of the Organism, the other sub-kingdom being Health. Disease must be defined for practical purposes as any departure from a more or less

theoretical state which we call health. The most constant and most important factor in health is the condition of the temperature, which is said to be healthy when it ranges at about 98.5° F. This standpoint has the advantage of bringing departures from health, and of bringing health itself, under the law of the Conservation of Energy. Any departure from this point upwards or downwards constitutes disease, and we have the first general division into diseases of elevated and diminished temperature. Properly speaking, the next question arising is whether the elevation or diminution of temperature is primary or secondary, because, in accordance with the law which has been formulated above, elevations of temperature are followed by depression, and depressions of temperature by elevation, before convalescence is reached. The next consideration is etiological. What is the cause of the elevated or diminished temperature? This cannot be overlooked, because elevation of temperature may be induced by cold or changing organic matter, but the state induced will have entirely different properties according to the cause. Causes divide themselves naturally into those acting from without and those acting from within—the exciting and predisposing of older writers. Causes acting from without may be inorganic or organic matter.¹ When inorganic matter acts *quá* matter, it induces such conditions as, e.g. Inflammation when it acts *quá* heat-abstracting, or Insolation when it acts *quá* heat-adding. When matter acts *quá* foreign it induces irritative inflammation, as, for instance, when a particle of sand sets up conjunctivitis. When it acts *quá* moving—when momentum is the essential factor in the action—Injury results. In the case of organic matter, this consideration is superfluous, since organic matter has no appreciable weight, and never acts in virtue of its momentum.² Organic matter in a state of change tends to set up its own changes in the organism, and it does so in

¹ It is not maintained that it is possible in the last analysis to differentiate inorganic from organic matter, but the broad division is tenable, and is convenient to work from.

² If a piece of wood, for example, inflicts an injury; here the essential factor is not the organised structure of the wood, but the momentum of the mass.

accordance with its own constitution. Thus when the constitution of the organic matter is of a low order, a specific inflammation results; when it is of a higher order, a fever results; and when it is of the highest of all, a parasitic disease ensues. The vanishing point between health and disease on this line is the act of impregnation and subsequent new development, the product of the ovum and spermatozoon being an example of organic matter in a state of change with the highest known tendency to specialisation. When we come to consider causes acting from within, our difficulties are immensely increased; but the general division into constitutional, diathetic, and hereditary diseases, in accordance with the definitions formerly given of these terms, will be found to hold, and to determine such conditions as gout, nervous susceptibility, instability of nervous equilibrium, cancer, etc., as well as various chronic affections of subacute intensity. Lastly, we have the conditions most difficult of all to class, in which it is not clear whether the cause is internal or external, but seems to be twofold, generally internal for the most part, but slightly due to external causes, such as a little cold or exposure or over-fatigue, which set up the latent predisposition to disease.

The diseases, therefore, which a sound classification should recognise, would seem to be:

1. Simple Inflammations, due to external inorganic matter acting as such, i.e. *quâ* heat-abstracting or heat-adding, or irritant, e.g. Cerebritis or Insolation or Irritative Inflammation.

2. Injuries, due to matter inorganic or organised, but essentially to matter *quâ* moving or having momentum. Examples are obvious.

3. Specific Inflammations, due to external organic matter in a state of change finding its way into the economy, and by virtue of what may be called selective affinity affecting some special portion of the nervous system, e.g. Hydrophobia.¹

¹ In the authoritative classification hydrophobia is placed among the functional nervous disorders, but the position assigned to it in the text seems to be far more consonant with its real character. The fact that it has been referred to functional disorders seems as good an instance as could be selected of the bad effects of functional names.

4. Fevers, due to external organic matter with a higher specialisation of nature than what causes the Specific Inflammations, e.g. Cerebro-spinal Fever.

5. Parasites, due to organic matter in the state of germ, or with the highest tendency to specialisation.

This exhausts the orders of diseases due to external causes, inorganic and organic. Nextly, we have diseases due to internal causes. A kind of intermediate Order is supplied by such conditions as have arisen from time to time in which foreign bodies have been introduced into the interior from without, and have thus acted as simple irritants. For example, such a case as that in which a man swallowed a fork; or a calculus in the bladder setting up inflammation there.

6. Diseases induced by eating improper food, either as regards quantity or quality, such as gout. Here the cause is internal organic, the food being first assimilated and then acting injuriously, for instance, by causing gouty deposit in the course of a nerve. Alcoholism should probably be classed here.

7. Such diseases as cancer, arising without apparent cause, e.g. in the optic nerve. Such a condition might be constitutional, or diathetic, or hereditary.

8. Diseases affecting the central nervous system, and induced by worry or anxiety or over-study, and culminating in such excitement as to induce Mania, etc., or in secondary Spanæmic Coma or Dementia.

So much for Orders. Genera are next determined by various considerations, as, e.g. the part affected, as Cerebritis, Meningitis, etc., Gouty Sciatica or Lumbago, Optic Cancer, and so on.

Species are determined by the characters of the disorder, whether acute or mild, chronic or brachychronic. And such varieties as strumous or suppurative would be usually found to depend on the nature of the constitution, of the diathesis or the heredity of the person attacked.

To take an example. Acute Cerebritis would be classed thus. Its order would be determined by its being due to inorganic matter acting *quâ* heat-abstracting, i.e. Inflam-

mation. Genus *Cerebritis*, depending on the part affected. Species *acute*, *brachychronic*, dependent chiefly on the constitution of the individual. Variety *simple*, dependent on healthy heredity or family history, or *strumous*, *suppurative* in variety, and *chronic*, *subacute* in species, dependent on unfavourable heredity or family history, or unfavourable individual history.

The impossibility of excluding a consideration of the causes of diseases in any attempt at classification has been already frequently adverted to, but I will finally refer to it now from another point of view. The course of disease seems to me to be essentially the same—the steps in its progress fundamentally similar, whatever be the cause. But the nature of the cause determines the nature of the affection. These steps are fundamentally three. First, there is depression of nervous power, e.g. as shown in the period of malaise ushering in fevers and inflammations, in the shock which is the first stage of the evil effects of injuries, or of the long-continued fatigue and depression ushering in chronic illness. Second, there is a congestive stage, otherwise called inflammatory fever, or fever proper, in which the temperature is high, and all the functions are excited. This seems to depend essentially on paralysis of the vaso-motor system of nerves. Lastly, there is a stage of depression of temperature below the normal, which invariably or all but invariably occurs before the normal is reached. Now these three stages seem to be common to all disorders whatever, and if they are looked at closely it will appear that they follow the law above formulated: that all agents capable of acting on the economy exert upon it contrary action in time. First, depression. Second, reaction, or congestion, or fever, if the action goes so far. Third, depression again. These three stages are common to all diseases, but the nature of the disease is determined by the nature of the cause. Hence the reason why a sound classification must found on etiology. How far we are still from a scientific classification is sufficiently obvious from the chaotic condition of medical literature, one writer following one plan and another another, while all mix up several plans together. It would be inte-

resting even to criticise the authoritative classification adopted by the Registrar-General if time and space permitted. At present it need only be said that it violates all the requisites of a sound classification, inasmuch as (1) it frequently repeats diseases; (2) it abounds in cross-divisions; (3) it repeatedly names as diseases what are really steps in the febrile and inflammatory processes; lastly, it makes no attempt to separate functional symptoms from disease-processes. The classification proposed in the text, it is contended, is free from these objections, and allows of all conditions that come under the notice of the medical man being referred without difficulty to their respective places, while the cardinal distinction between function and physical change has been kept in view throughout.

CALORIMETRIC OBSERVATIONS
UPON THE
INFLUENCE OF VARIOUS ALKALOIDS
ON THE
GENERATION OF ANIMAL HEAT.

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THE following experiments on the modifying influence on temperature of some of our more potent drugs were undertaken from the conviction that more exact knowledge is requisite on these points ; and also that preconceived opinions might be tested by the apparatus which the practical physiologist places at our service, and their accuracy thus be confirmed or refuted ; and lastly, as a matter of equal importance, that work already done in this field should be subjected to further and fuller investigation by repeated and frequent observations. In dealing with the subject I shall endeavour to confine myself almost exclusively to facts, stating plainly the results of actual experiment. More especially is this necessary at the present period, when the whole subject of animal heat appears to be undergoing thorough reviewal : this is, in fact, a transition period, which fairly bids to erase many old and preconceived views with regard to the development and regulation of animal heat. It is a noteworthy fact that even Claude Bernard, to whom we are indebted for first drawing the attention of physiologists to the Great Sympathetic System, as embracing amongst its most important

functions a regulative jurisdiction over animal temperature, fairly admits himself a convert to the views of Schiff and Goltz in his new and most interesting work on Animal Heat.¹ 'Y a-t-il, en un mot, des nerfs *constricteurs vasculaires*? Je réponds sans hésitation par l'affirmative.' At the same time Bernard freely admits the anatomical objection which occurs to all of us with regard to a mechanism of *active* dilatation. 'L'existence d'une couche musculaire composée de fibres transversales, innervées par le grand Sympathique, et se contractant sous l'excitation de ce nerf, permet de comprendre le resserrement du vaisseau. Mais le phénomène de la dilatation est actuellement tout à fait inexplicable' (Op. cit. p. 232). Prior to entering directly upon the subject in hand, it may be well to explain briefly the method adopted in these investigations, and to describe shortly the calorimeter employed in estimating the amount of heat set free from the body.

The Calorimeter.—The instrument made use of by me in other investigations, an improved form of the older one, was suggested by Dr. Burdon Sanderson, and made by Mr. Hawksley of Blenheim Street. A description of the older form of instrument may be found in the 'Handbook to the Physiological Laboratory,' but I regard the new calorimeter not only as a more perfect instrument, but less complicated, and in my own experiments I must say it has afforded every satisfaction. This instrument consists of two boxes or chambers of tinned sheet iron, an inner and outer one. The inner or smaller chamber is surrounded on all sides (except above) by water which is contained in the outer box, and on which it really floats. The measurements of the inner and outer compartments are respectively as follows: length, 18 and 22 inches; breadth, 10 and 14 inches; depth, 11 and 15 inches. Around the upper border of the inner chamber runs a shallow gutter, into which the rim of the lid is received, and which, containing water, shuts off effectually all communication between the air in the chamber and the surrounding atmosphere, except at two points, where exit tubes of copper arise, one from the cover and the other from

¹ 'Leçons sur la Chaleur Animale.' 1876.

one side near the base of the compartment, the latter being 17 inches in length. By this means thorough condensation of the pulmonary and cutaneous exhalations is ensured, and little or no loss of heat from this source need be feared. The lid of the inner compartment is secured firmly by bolts, and when this box is fixed by straps within the outer casing it is found to be separated from the latter below and on all sides by a clear interval of two inches; this space is filled up with water, the weight of which must be known. The lid of the outer casing is perforated for the passage through it of the two copper tubes before alluded to. Passing diagonally across the *exterior* of one of its sides, from the upper to the lower angle, is a substantial copper tube of much greater calibre than those connected with the inner box, and which opens into this outer chamber at its upper and lower extremity, and becomes necessarily filled with water from the same compartment. Being external to the box itself it is kept gently warmed by a small gas jet placed below it, and by this means a steady current of the fluid is kept up, and an equable and fixed temperature can be ensured. We thus dispense with the third casing of wood which was used in the older instrument, and which, being filled with tow, was employed for excluding fallacies arising from radiation or conduction of heat.

Theory of its Application.—With this brief description of the calorimeter it will be readily understood that the amount of heat set free from the surface of the animal in the inner chamber is retained by the total bulk of metal and fluid included in the calorimeter, and that the rise in temperature of the water enables us to compute the actual amount of heat thus given off. But as we are chiefly concerned with the total heat-*formation* in a given time, it becomes requisite to eliminate an element which would otherwise be a source of fallacy: this is the retention of part of this new heat-formation by the animal's body, or, on the other hand, loss of heat from its own body temperature. To be more explicit: should the heat-formation be equal to the amount set free from the body, the temperature of the animal will remain constant; should the temperature of the animal become

elevated, it is apparent that a portion of the heat generated must have been retained and appropriated in raising the animal's temperature, an amount which must not be lost sight of, but which must be estimated together with the amount set free from the surface of the body, and which is retained by the calorimeter. On the other hand, a *fall* in the animal's temperature indicates to us that the temperature of the calorimeter does not express alone the amount of heat generated, but this amount *plus* a quantity borrowed directly from the animal's temperature. Four thermometrical readings are therefore requisite for each calorimetric observation as follows :

1. Temperature of animal when placed in the calorimeter.
2. Temperature of calorimeter at this same period.
3. Temperature of the animal upon removal from the calorimeter.
4. Temperature of calorimeter at this latter period.

Calorimetric observations have of course nothing to do with the actual amount of heat present in the body at any given time, but simply deal with the new formation generated to supply the loss constantly occurring, and therefore directly involve the balance maintained between the heat-discharge and heat-supply. On the other hand, thermometry has as its scope the existing temperature of the body, and its alternations of increase and decrease compared with certain assumed standards, as the zero of Reaumur, Celsius, or Fahrenheit. Thus if our thermometer registers the elevation of the animal's temperature as 1° C., it simply informs us of an elevation of temperature as compared with the zero point of the centigrade scale, and affords us no clue as to whether this elevation is due to increased heat-formation, decreased heat-discharge, or both conditions. In either case the result must be the same from information conveyed by our thermometer. It is on these points that calorimetry steps in to our assistance, and affords the desired information, and to the value of each of these individual points I shall have to return later and shall endeavour to indicate their significance. Meanwhile, I must deal with the method to be pursued in obtaining an accurate calorimetric observation.

Calorimetric Calculations.—We must first obtain the

thermal value of our calorimeter, in other words, the number of gramme-units of heat requisite to raise the whole calorimeter one degree of the centigrade scale.¹ The amount of heat expended in raising the temperature of any substance one degree is calculated readily in gramme-units by multiplying the specific heat of the substance by its weight in grammes. Hence our calorimeter, consisting, as it does, of diverse metals as well as fluid, the specific heat for each must be multiplied by their individual weights, and the various products combined express the number of gramme-units of heat required to raise the calorimeter one degree of the centigrade scale. The same calculation must be next applied to the animal, that is to say, its specific heat must be multiplied into its weight in grammes. The specific heat of an animal body is estimated at 0·83, so that for an animal weighing 2000 grammes, 1660 gramme-units of heat would be necessary to raise the animal's body one degree in temperature.

The following experiment upon the effects of ergotine administered to a rabbit by subcutaneous injection, and given in full detail, will suffice to explain each step taken in a calorimetric observation. It is presumed that the value in heat-units of the calorimeter has been previously estimated, and the following data obtained:—Weight of animal = 1675·613 grms. Heat-units required to raise calorimeter 1° C. = 46667·2187. Heat units required to raise animal 1° C. (1675·613 × 0·83) = 1390·7587. Temperature of calorimeter before experiment = 15·55° C. Temperature of animal before experiment = 39·44° C.

Injected 10 grains of Ergotine.

Time	Temperatures	Variation	Equivalents in heat-units	Heat generated
$\frac{1}{4}$ hour {	Calorimeter = 15·55° C. Animal = 39·38° C.	Nil Loses ·06° C.	∞ 83·4455	...
$\frac{1}{4}$ hour {	Calorimeter = 15·83° C. Animal = 39·13° C.	Gains ·28° C. Loses ·25° C.	13066·8203 347·6896	12719·1307
$\frac{1}{4}$ hour {	Calorimeter = 16·11° C. Animal = 39·5° C.	Gains ·28° C. Gains ·37° C.	13066·5203 514·5807	13581·4010

¹ It is hardly necessary to remind the reader that a gramme-unit of heat signifies the amount consumed in raising one gramme of water 1° C.

The foregoing experiment involves, therefore, three observations, each a quarter of an hour in duration. The first interval affords us either evidence of almost entire suspension of heat-formation, or else of arrest of the loss from the surface of the body to the calorimeter, and involves a question of great interest which must be deferred for subsequent consideration. That the loss of 83·4455 heat-units from the animal's temperature causes no appreciable variation in the temperature of the calorimeter is readily understood, as it could but raise the whole the 1·559th of a degree centigrade, a fraction far beyond the limits of our most delicate thermometers.

In the two last intervals over 12,000 and 13,000 gramme-units of heat respectively are generated. A very large amount, and which divided by the animal's weight in grammes, gives us 7·59 and 8·1 heat-units, generated for every gramme of body-weight. This latter relationship of heat-production to weight is a convenient form of expression, and will be retained in each future observation. In the following notes of calorimetric observations I have selected *typical* results from a large number of experiments, including only such results as were confirmed by frequent repetition of the experiment. The drugs whose action upon the thermogenetic functions of the body it was thought advisable to examine were as follows :—

Atropine.	Picrotoxine.
Solanine.	Ergotine.
Hyoscyamine.	Chloral.
Strychnine.	

Having premised so far, I will take the series in the order above given, commencing with calorimetric observations upon the actions of atropine.

ATROPINE.

The physiological action of atropia depends to a great extent upon the dose administered, the effects of a medicinal dose being entirely different from that of a lethal dose. The

perusal of the foregoing experiments (table A.) will, I believe, clearly convince us that two distinct and well-defined periods succeed the administration of toxic doses of atropia. The first period is marked by dilatation of the arterioles, the ears becoming hot and engorged, and a great increase in tissue-change is evidenced by the large amount of heat generated together with a *fall* in the animal's temperature, the latter due undoubtedly to the dilated state of the peripheral vascular system, whereby the dispersion of heat is greatly favoured.

Comparison of these results with those obtained from animals in a normal condition will at once indicate the great divergence from the healthy, regulative administration of the vaso-motor centres. Here we have over 12 kilogramme-units of heat produced in the space of fifteen minutes, which amount to 6 and even 8 gramme-units of heat for each gramme of the animal's weight. Nor must this be regarded as due, in any great measure, to direct *abstraction* from the normal temperature of the animal, although there was a decided fall, which in one case amounted to 1.59° C. The amount, however, from this source forms but a trifling item of the large quantity generated afresh in the tissues.

Following this primary toxic-depressant action comes the second period alluded to, in which the former conditions are entirely reversed. In this stage we find the heat-production greatly lessened, the little given off being frequently not appreciable by the calorimeter. This profound change evidently points to universal spasm of the capillaries, and the diminished tissue-change consequent thereupon, in its turn attributable to stimulation of vaso-motor centres.

Here also we get *retention* of heat; just as in the primary stage we found a loss going on from the body-temperature, so now does the animal's temperature slowly but steadily rise.

Lethal doses of atropia have been observed by most writers to produce a rapid fall in blood-pressure, as a result of capillary dilatation from the direct action of the alkaloid on their coats, and as Bezold and Bloebaum have shown, by

its direct depressant action on the cardiac muscle, a fall in temperature has been also noticed; we may now assert from the above cases that this *fall* in temperature is coincident with a greatly increased heat-formation.

In the 3rd experiment (Table A.) we observe the effects of smaller doses of the alkaloid, the direct results of their injection being an increased heat-formation together with *retention*, so that the animal's temperature becomes raised; a febrile condition is thus produced which may be maintained for a long period by employing minute doses, and also by frequent administration. Thus the primary stimulant effect of the 3-grain injection gave place to general arterial dilatation and fall of temperature, but the original state was immediately re-established upon a second injection of 5 grains of the alkaloid.

SOLANINE.

The effects of this alkaloid are most pronounced upon thermogenesis, and its action on the vaso-motor regulative system favours *retention* of heat, the animal gradually rising in temperature. The subcutaneous injection of 2 grains caused a persistent elevation of temperature which had in one hour and a half reached 3.1° F. (1.72° C.) above the normal. In several cases, however, the injection of larger doses produces a primary but very temporary fall, followed invariably by the gradual elevation of temperature, tumultuous cardiac action, and extremely hurried breathing. During this febrile period the amount of heat generated and acquired by the calorimeter has fallen to zero, in fact is not appreciable by the thermometer.

There are more explanations than one to the rise of body-temperature; an elevation may be due to a preponderance of heat-formation over the amount of heat-dispersion. Again the thermal discharge may be diminished, the generation of heat being normal in amount; and here again we should get an elevation of temperature without actual increase of heat-formation. Now is this latter condition the one explanatory of the action of solanine? Decidedly not; for though heat-

elimination is most certainly reduced to a minimum, if the amount generated maintained its normal standard, and was retained *wholly* and added to the body-temperature, we should have obtained a rise far more rapid and intense than the one observed. A momentary consideration of the large amount of heat eliminated in the natural condition every half hour will prove to us how untenable such a proposition must be. In fact we have here not alone a *retention* of heat but, a most marked *decrease* in thermogenesis.

Reference to the table of experiments with solanine seems to indicate a return to the initial condition through a stage of large heat-formation and increased dispersion from the body-temperature. Thus in Experiment 1 (Table B.), after three successive periods of arrested heat-formation we obtain a sudden registry by the calorimeter of nearly 14 kilogramme heat-units per quarter hour, equivalent to 8.4 heat-units for every gramme of the animal's weight; thence through a period of declining temperature thermogenetic activity reaches the normal standard. I need not dilate further here upon the physiological action of solanine, which has yet to be worked out fully, my object in this article being merely to draw attention to the main facts observed in calorimetric observations upon the employment of these toxic agents.

HYOSCYAMINE.

This potent alkaloid, belonging to the plants of the same natural order as those we have just considered, shares with them the property of modifying thermogenesis to a very great extent. The administration of large doses is accompanied by a great thermic discharge, the calorimeter rapidly rising in temperature. By far the larger portion of heat thus set free depends upon increased heat-formation, although a small portion is directly abstracted from the animal's temperature, which consequently falls to a greater or less extent. An early primary effect of the alkaloid extending, however, over only a *short* interval, is often observed. In this period the temperature may fall slightly, and the actual

formation of heat be diminished instead of being increased, or the amount generated may remain normal, whilst a slight fall in body-temperature ensues. This *diminished* thermic discharge, noticed in the earlier action of larger doses, is still better marked and usually more protracted when smaller quantities of the alkaloid are administered, and is evidently due to stimulation of the sympathetic. The physiological action of hyoscyamine has been very fully worked out by Dr. Robert Lawson of the West Riding Asylum, and the calorimetric observations made by myself entirely confirm the results obtained by Dr. Lawson. In his article on the 'Physiological Actions of Hyoscyamine' in the 'West Riding Asylum Reports for 1875,' the writer states emphatically that there is a temporary stimulation of the sympathetic preceding the paralysing action of the drug upon the par-vagus, and that the temperature modifications are lessened as the dose is decreased. On referring to our table again it will be observed that a small dose of half a grain was succeeded by increase of temperature to a very slight extent, whilst a grain and a half was succeeded by neither rise nor fall of body-temperature (*vide* Experiment 4). In these latter experiments rabbits were used whose cervical sympathetics had been cut on both sides and the ganglia extirpated. Now in these rabbits for many days succeeding perfect union and healing of the part operated upon, calorimetric observations showed a large amount of tissue-change, and as a result an enormous thermic discharge amounting to 12·5 gramme-units for every gramme of body-weight, in half-an-hour's observation. This animal, therefore, whose heat set free was over 14 kilogramme-units in each interval of half-an-hour, i.e. 7 kilogramme-units for each quarter-of-an-hour, threw off in the latter time but 4 kilogramme-units after the injection of half-a-grain; and only 2966 gramme-units after the administration of a grain and a half. Occasional variations occur in the action of hyoscyamine on the rabbit, the cause of which I have not been able to appreciate. The third experiment in Table C. will serve to illustrate these exceptional cases. The primary effect here was altogether unusual, and the sudden elevation of temperature from *retention* of heat

throughout was remarkable. The high rate of heat-generation commencing at 19 kilogramme-units, and falling rapidly to an amount quite inappreciable, and again suddenly increased to 32 kilogramme-units, was a phenomenon I had never before met with in hyoscyamine poisoning, and one which I certainly cannot comprehend.

STRYCHNINE.

Calorimetric studies of the action of the excito-motor drugs strychnine and picrotoxine exhibit a marked resemblance in effects on animal heat. In both the vaso-motor disturbance is profound, and in both do we observe a primary stage of this disturbance, introducing us to the succeeding and more purely toxic symptoms.

Reference to the first experiment under the head of Strychnine (Table G.) will exhibit clearly the general features of the primary stage referred to, the dose being too small to induce any serious symptoms and no motor derangements.

In the rabbit operated upon, the $\frac{1}{180}$ th of a grain only being injected, quarter-hour observations gave five, two and three kilogramme-units of heat as the amount actually generated, and comparing this with the weight of the animal, we found it corresponds to over five, two and three gramme-units of heat respectively to every gramme of body-weight—a large amount to be furnished in so short a period. The primary effect, then, of strychnine is a general increase of heat-formation; but this stage is especially prolonged when minute doses are administered, the effect of poisonous doses being to greatly shorten the duration of this period, and to usher in rapidly the symptoms characteristic of a toxic dose. The second experiment in the same table serves well to illustrate the action of a fatal dose of strychnine. The dose administered was the $\frac{1}{96}$ th of a grain; the rabbit weighed 1197 grammes, the dose, therefore, being so apportioned as to have a certainly fatal effect. This result, however, was to be combated by its antagonist, chloral, administered in sufficient quantity to save the animal. For this purpose, after an elapse of ten minutes from the strychnia injection,

sixteen grains of chloral were given hypodermically. The temporary increase of thermogenesis here is rapidly followed by a reversal of these conditions. The amount of heat thrown off being inappreciable for three intervals of quarter of an hour each, and during which period a fall in the animal's temperature occurred. This extreme depression of functional activity resulting in so small an amount of heat-formation must have been explicable only on the supposition of a very *general* and *extreme* spasm of the capillaries, resulting likewise in a depression of the body-temperature 1.7° C. (3.1° Fah.). During this period the reflex excitability was greatly increased, the slightest touch or blowing on the animal giving rise to spasmodic starts. Three quarters of an hour after administration of chloral the animal when removed from the calorimeter was thoroughly under the influence of chloral, the pupils dilated and insensible to a brilliant light. The convulsive twitchings on the slightest touch still occurred. It was at this period that chloral was exerting its antagonistic properties fully, and as a result of this action we found above eight kilogramme-units of heat generated at intervals of a quarter of an hour, or to be exact, 6.7 and 7.2 gramme-units for each gramme of body-weight. The reduction in temperature of the animal had also amounted to 3.95° C. (7.1° Fah.) since the administration of the chloral. The animal recovered perfectly from the action of those drugs. With regard to the general arrest of heat-formation on the administration of strychnine, it must be noted that Richter and Mayer have testified to a general vaso-motor spasm; and that this is independent of convulsions is pointed out by Wood, who states that the rise in arterial pressure occurs in curarised animals after strychnine has been taken. It is evident, therefore, that prior to the muscular tetanus a vascular tetanus ensues; in other words, that strychnine acts first upon those spinal centres from whence the vaso-motor constrictors proceed. This extensive arterial spasm represents so much work done; but it must also be remembered that the universal dilatation of the vessels after the physiological action of the chloral was well established, is attended with great increase of tissue-change, and the work thus

accomplished is chiefly represented by the heat evolved at this time. In fact we have here substituted for a state of tetanised muscles and vessels, a universal vascular dilatation (active perhaps), admitting of active chemical changes in the tissues, and as a consequence an immense evolution of heat. To me it appears but the substitution of one species of work for another. In all convulsive attacks, whether such as are induced by toxic agents, such as strychnine and picrotoxine, or those affections of the motor cells significant of abnormal charging to excess with potential energy resulting in convulsive discharges, the passage from a labile to a more stable equilibrium by remedial agents appears always to depend upon vaso-motor changes. I think it highly probable that in such cases we simply divert the discharge into another channel, and the heat-discharge thus induced may be regarded as a correlation of the motor-discharge from the spinal centres.

The fall of temperature induced by chloral is a well-recognised fact; when, therefore, it is administered in cases of epilepsy, where from the existence of an aura we recognise the advent of an attack, the arrest of this convulsive seizure is almost invariably attended by progressive elevation of temperature, a condition wholly opposed to the usual action of the drug. I can but explain it as a substitution of thermal for motor discharges from the central nervous system. To this fact I have already endeavoured to draw attention in the records of a case of peculiar interest bearing upon this question.¹

PICROTOXINE.

The great resemblance between the convulsions produced by this potent drug and those observed in epilepsies is too well known to be more than merely alluded to here. The primary effect of administering a large dose of picrotoxine appears to be the generation of an enormous amount of heat reaching from 12,000 to 48,000 gramme-units per half-hour, i.e. from 10 to 38 heat-units for each gramme of the animal's

¹ Vide 'Medical Times and Gazette,' 1875.

weight. This period, therefore, coincides with that of the primary stage of strychnine poisoning, but is far more prolonged, as it frequently extends over an interval of half-an-hour to an hour when large doses of the poison are administered. Then ensues a marked spasm of the capillaries, during which the heat-production is reduced to a minimum, a stage which immediately precedes the convulsive outburst. It will be observed on reference to Table E., that in the second and more characteristic case where the dose was so proportioned to the weight of the animal as to produce slight general convulsions without a fatal effect, this period of excitation of the vaso-motor constrictors existed for full half an hour, was succeeded by intervals in which there were marked alternations in the thermogenetic results, varying between two, eight, and twenty-one kilogramme-units, at intervals of thirty minutes. This latter period coincided with the convulsive seizures, and was attended with a slight rise in the animal's temperature, contrasting strongly with the primary stage preceding the outburst of convulsions, during which there was a fall of fully 5.6° Fah., (3.20° C.) in the temperature of the body.

This rise of temperature during the onset of convulsion amounted, however, at furthest to but 1.2° Fah. ($.77^{\circ}$ C.), and was quickly succeeded by a fall of nearly 2° Fah. That it was due to a temporary vascular dilatation is confirmed by the statement I find I have appended to my notes of the case at this period, where it is said, 'at this period the ears were congested and hot, and on being compared with a rabbit, the superior cervical ganglia of which had been extirpated, the vascular engorgement quite coincided with it in degree.' Agents which interfere with this extensive vascular spasm have been found the most useful in combating the poisonous symptoms of picrotoxine, and the antagonism existing between chloral and this principle has been satisfactorily established by Dr. Crichton Browne, and the remarks made with regard to its action in strychnine poisoning are equally applicable here. Before dismissing the subject of picrotoxine poisoning I would wish to draw attention to one fact which I consider of importance. By

some physiologists and prominently amongst these by Mosso, the movements of the pupils have been regarded as produced by a species of hydraulic mechanism dependent upon vascular turgescence or the opposite condition—in fact a peculiar erectile property is stated to account for pupillary movements. Even if this be the case we do not eliminate from the question the nervous supply upon which dilatation is supposed to depend; the vascular arrangement of course is under the direct control of the vaso-motor centres. How then is the following result explained?

A rabbit in which both superior cervical ganglia of the sympathetic had been extirpated, and which exhibited in a marked degree all the concomitant symptoms, was given a dose of picrotoxine just sufficient to produce convulsions. On several occasions the onset of the convulsion was accompanied by a *wide dilatation of the pupil* followed by a contraction to its former dimensions. At the onset also in a thermometer placed in the animal's ear, the mercury standing at 102·4° Fah. fell suddenly to 98·6° Fah. from the excessive vaso-motor spasm. The latter fact proves that picrotoxine acts directly upon the vessels through their muscular coats.

ERGOTINE.

The powerful influence which this drug exerts upon the circulating system induced me to study its effects upon the production and dispersion of animal heat. The primary action is so suddenly induced that it was found necessary to take observations extending only over periods of a quarter of an hour. In all cases, as evidenced by the appended experiments (*vide* Table F.), we get as the first result a sudden and profound arrest of thermogenetic activity, during which period also there is a decided fall in the animal's temperature. A vessel in the ear which measured ·9 mm. in diameter, was reduced to ·5 mm. almost immediately after injection of the ergotine. Succeeding to this stage of vascular spasm, we get an increase of tissue-change evidenced by the formation of six, eight, and, in one case, thirteen

heat-units for each gramme of weight, and a progressive elevation of temperature amounting in the first experiment (Table F.) to 1.09° C. (2° Fah.). This retention of heat resulting in elevation of temperature, evidently depends on persistent spasm of the cutaneous capillaries, which gradually passes off, or to the preponderance of heat-formation over thermic discharge; thus in the second experiment just alluded to, the increase of temperature depends upon the successive additions of gradually lessened increments of heat to the animal's temperature, ranging from 514 to 76 heat-units per 15 minutes. Now do these data agree with the facts ascertained with regard to the physiological action of ergotine? It must be remembered that Dr. Holmes¹ has published the results of twenty-three experiments performed with ergotine upon animals, and although the kymographion was employed, the most conflicting and diverse results ensued, leading Dr. Holmes eventually to the conclusion that the primary effect of ergotine was an instantaneous fall in arterial pressure. This was of course directly adverse to preconceived opinions, and has apparently been disproved by the most elaborate experiments of Ebertz performed at a later date.² He invariably obtained as his result, an instantaneous and enormous rise of arterial pressure, and attributes it to vaso-motor constriction. The results of my experiments rather tend to confirm the views of Brown-Séquard,³ who insists upon two periods in ergotine poisoning; first, vaso-motor spasm; secondly, vaso-motor paralysis. The large amount of heat-formation following upon the primary spasm of the vessels I incline to refer to vascular dilatation of a more or less general character, and the elevation of temperature to a preponderance of this formation over its discharge, regulated by the state of pulmonic and cutaneous capillaries. Should this be proved to be the case, it will become of import where ergotine is administered to restrain epistaxis and other hæmorrhages, as also the colliquative perspirations of disease.

¹ 'Archives de Physiologie,' t. iii. 1870.

² 'Inaug. Dissert.,' Halle, 1873.

³ 'Archives de Physiologie,' t. iii. 1870.

CHLORAL.

Upon the administration of large doses of chloral to rabbits, observations extending over periods of thirty minutes invariably gave results similar to the first experiment tabulated under the head of this drug (Table D.). A great increase of heat-formation ensues, accompanied by a large evolution also from the animal's temperature, which falls fully 6.3° Fah., represented by 2016 heat-units, in the period of one hour. Hammersten's statement that this rapid fall of temperature is dependent upon diminished heat-production (as he states it occurs when animals are wrapped up and laid in a warm place), is, I consider, fallacious; in fact all my observations tend to confirm the statement previously made, viz., that the heat-production is greatly increased, and that the fall of temperature is really dependent upon the increased dispersion of heat from the body ensuing from exposure during very general vascular dilatation. Hence the absolute necessity in cases of chloral poisoning of keeping our patient warmly wrapped, so as to reduce this surface-loss to a minimum, a procedure the value of which is substantiated by the experience of most observers, and to which I can bear personal testimony. On reference to the succeeding experiments in the same Table, a primary condition will be observed, where there is an actual deficient heat-production. When a large dose is exhibited, such as in Experiment 2, an observation must be made within a short interval to clearly appreciate this initial result of chloral. Thus in fifteen minutes interval the amount generated was scarcely at all appreciable, nor was it due to *retention*, for the body-temperature had fallen.

Again, in Experiment 3, this period is greatly extended in duration—an apparently anomalous action, really, however, due to the small dose administered, the resulting thermal increments being far less marked than in the preceding experiments. This result tends to confirm the opinions of Anstie, Andrews, and Bouchat, that sphygmographic tracings indicate a primary increased arterial

tension when small doses are administered, and which is attributed to capillary contraction.

Eventually, however, we obtain in all these cases a general vaso-motor dilatation and fall of blood-pressure as a result of this and the cardiac implication. I have thought repeatedly that I could detect a stage of slight pallor precede in man the stage of hyperæmia, and in rabbits I feel convinced of having witnessed a diminution in the calibre of the blood-vessels of the ear succeed to the administration of chloral as a primary result.

TABLE A.—ATROPINE.

(Observations extending over 15 Minutes.)

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
12 grains	12701·4056	...	365·4147	7·5 heat-units
	*	...	316·2243	*
	*	210·8162	...	*
	*	...	70·2720	*
	6455·3917	38·6496	...	3·8
	1795·9686	115·9489	...	1·05
<i>Obs.</i> —The asterisk (*) indicates that the amount of heat communicated to the Calorimeter was so small as to be inappreciable.				
5 grains	12735·6516	...	400·1695	8·55
	5879·4194	349·3543	...	3·6
	612·2043	67·7535	...	·4
<i>Obs.</i> —The first two lines indicate the quantity generated during $\frac{1}{4}$ hour intervals, these results being maintained upwards of 1 hour.				
3 grains	6650·8571	117·4470	...	4·23
	12234·7573	...	365·3909	7·7
5 grains again injected	13288·6646	221·8444	...	8·4
	*	...	443·6888	*
10 grains	12143·2473	...	550·2353	6·5
	6409·4135	...	123·9966	3·4
	*	123·9966	...	*

TABLE B.—SOLANINE.

(Observations extending over 15 Minutes.)

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
2 grains	2951·6799	151·6470	...	1·7 heat-units
	*	386·0105	...	*
	*	356·0105	...	*
	*	454·9410	...	*
	13990·4883	923·6681	...	8·4
	13135·7507	68·9304	...	7·9
	*	...	689·3046	*
	1808·6934	...	212·5525	1·08
	3172·5497	...	94·1568	1·9
$\frac{1}{2}$ grain	19492·0773	358·5189	...	10·3
	*	...	233·8166	*
	About the same
	for the follow-
	ing 3 observa-
	tions
3 grains	*	...	851·6219	*
	*	*
	12736·1487	...	237·3372	7·5
	*	544·4795	...	*
	*	237·3372	...	*
5 grains	25154·3626	...	512·6060	13·6
	*	596·7652	...	*
	*	512·6060	...	*
	10729·6773	696·2260	...	5·8
	*	76·5083	...	*

TABLE C.—HYOSCYAMINE.

(Observations extending over 15 Minutes.)

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
3 grains	2398·3157	...	51·7130	1·425 heat-units
	3498·9448	...	234·4324	2·1
	5034·5115	537·8155	...	3·02

TABLE C.—HYOSCYAMINE—*continued*.

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
4 grains	9402.0179	...	530.7964	4.95
	9402.0179	...	530.7964	4.95
	15516.8492	8.15
3 grains	19965.6214	365.3909	...	12.6
	7107.5958	574.1857	...	4.5
	*	78.298049
	32512.0129	78.2980	...	20.6
$\frac{1}{2}$ grain	4150.0370	52.9427	...	3.65
Went on at this rate for two hours.				
$1\frac{1}{2}$ grains	2966.7009	1.25
<i>Obs.</i> —This rabbit had both superior cervical ganglia of the Sympathetic extirpated a few weeks previously, and gave as a result <i>without</i> drugs:—				
	7084.9377	82.3554	...	6.25

TABLE D.—CHLORAL.

(Observations extending over 30 Minutes.)

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
20 grains	10815.7766	...	1008.8788	7.7
<i>Obs.</i> —At this for one hour, and died narcotised.				
17 grains	*	...	542.1722	*
	16907.9614	...	825.5804	11.3
	15344.6226	...	1922.2471	10.3
<i>Obs.</i> —The last two lines represent half-hour intervals.				
10 grains	4870.2005	...	453.1002	1.4
Continued thus } over 2 obs.	2588.6132	...	116.1793	.75
The same for 2 } obs.	9510.2485	...	1068.8197	5.6

TABLE E.—PICROTOXINE.

(Observations extending over 30 Minutes.)

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
$\frac{1}{20}$ th grain	9099·1092	465·6743	...	7·27
	48230·5674	Nil	Nil	38·5
	At this rate } for 1 hour }	Nil	Nil	...
Obs. Convulsions } began }	8438·4076	...	1561·71	7·9
	*	...	2405·4834	*
$\frac{1}{20}$ th grain	12677·0724	...	1323·0922	10·5
	17279·6535	...	1387·2327	14·3
	25065·5631	...	601·4055	20·8
Observations	*	...	1804·2166	*
Slight Convulsions	2402·2317	902·1083	...	1·99
General Convul- } sions }	21488·8854	488·6384	...	17·8
General Convul- } sions }	8515·5252	...	817·9115	7·0

TABLE F.—ERGOTINE.

(Observations extending over 15 Minutes.)

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
10 grains	*	...	83·4455	*
	13414·510	...	347·6896	8
	13581·4011	514·5807	...	8
	3454·4575	375·5048	...	2·05
10 grains more } injected at this } period }	12600·1482	Nil	Nil	7·5
	2003·1258	236·4289	...	1·65
	776·4999	76·4917	...	·46
15 grains	*	...	435·6291	*
	24391·1241	Nil	Nil	13·4
	10531·2765	132·2445	...	5·6
	12677·4391	38·8954	...	6·7
	7062·3884	264·4891	...	3·7
20 grains	*	...	896·0235	*
	12810·7836	...	256·0367	6·6
	*	976·0256	...	·5
	6609·4148	176·0046	...	3·4

TABLE G.—STRYCHNINE.

(Observations extending over 15 Minutes.)

Dose	Heat-units generated	Retained by body	Lost	Amount formed per gramme of body-weight
$\frac{1}{180}$ th of a grain	5215·1168	630·1183	...	5·46
	2224·1373	...	451·7829	2·32
	3001·6649	...	63·4081	3·14
	3266·7050	Nil	Nil	3·42
<i>Obs.</i> —No convulsive twitching or other symptom of strychnine poisoning.				
$\frac{1}{96}$ th of a grain 16 grains of chloral	6284·6836	...	701·9827	5·25
	*	...	265·8530	*
	*	...	745·293	*
	*	...	24·8431	*
	4033·0633	...	828·105	3·35
	4318·9174	...	346·8041	3·6
	*	...	629·3598	*
	*	...	178·8706	*
$\frac{1}{120}$ th of a grain	13309·6480	242·8277	...	7·7
	*	157·1238	...	*
	3502·3907	235·6857	...	2·
	*	199·9757	...	*
	*	314·2476	...	*

HYOSCYAMINE IN THE TREATMENT OF SOME DISEASES OF THE INSANE.

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IN the last volume of these Reports, and in the number of the 'Practitioner' for July 1876, I had the opportunity of publishing several observations regarding the properties of hyoscyamine. In the first paper I confined myself exclusively to a statement based on a very prolonged observation of the physiological actions of the drug on men and animals, and in the second I detailed several instances in which hyoscyamine had been of clinical value, and made passing remarks specifying in what diseases the drug would probably be useful, and in what prejudicial. With regard to the physiological actions of the alkaloid, it was determined that the effect on man of a moderate dose was the production of a mental condition which partook of all the leading symptoms of simple mania, but which, in addition, was characterised by extreme physical helplessness, intermittent drowsiness, hypermetropia and dryness of the mouth, lips, and throat. With regard to the therapeutic properties of the drug, I started with the principle that the succussion produced in the mind already muddled with delusions and hallucinations, by the antagonism of new forms of mental aberration, together with the subsequent deep and prolonged sleep produced by hyoscyamine, might tend to leave the

mental state more composed after the artificially-produced delusions and hallucinations had passed away. In the paper published in the July number of the 'Practitioner' I have adduced instances proving that, whether that principle is right or wrong, the drug does possess properties which render it potent in the removal of insane conditions over which drugs more exclusively narcotic have little or no influence. At that time I confined my attention almost entirely to the demonstration of the value of hyoscyamine in the treatment of sub-acute recurrent and chronic forms of mania, more especially such varieties as are associated with delusions of suspicion, laying particular stress on the advantages derived from the use of the drug amongst such patients as crowd the refractory wards of English asylums, in whom mania sometimes appears to be the result of an inherent moral baseness, the exercise of which has not unfrequently rendered the patient an unwelcome and, latterly, a discarded member of the rank and file of the British army, has submitted him unsuccessfully to the repeated discipline of numerous prisons, and finally has brought him under the protecting roof of a lunatic asylum, where, being fully conscious of the partial immunity from punishment which his certificate and position confer upon him, he revels in the performance of wanton mischief. I brought forward at least one instance in which a worthless, and worse than worthless, specimen of this class had been reclaimed to industry by the use of hyoscyamine, and since then there have been frequent opportunities of noticing in this institution that the physical powerlessness produced by hyoscyamine has a moral influence in leading such patients to control the voluntary part of their excitement, while the sedative action of the drug allays, for a time at least, the outbreaks of functional or organic irregularities. Hitherto I have spoken only of the use of hyoscyamine in single doses; I have now to supplement what I have written of the drug under that head, and to refer to its employment in smaller quantities administered continuously.

In speaking of the treatment of several forms of insanity by single doses sufficient to produce the physiological actions

of the medicine, I shall have to record further instances of its use in Mania with delusions of suspicion, and in simple and recurrent Mania, but I shall take notice of the efficacy of the drug in the Epileptic Status, in Mania associated with logorrhœa, or incessant incoherent talking, and in insane conditions characterised as a prominent feature by constant and apparently involuntary, or at least incontrollable, destructiveness.

In speaking first of the employment of hyoscyamine in the treatment of the Epileptic Status, it is necessary to observe that under ordinary circumstances, and in the great majority of instances, hydrate of chloral is so absolutely certain to arrest the progress of the fits which in the aggregate make up the status, that hyoscyamine is rarely required for the production of that happy result. In the actual practice of this Asylum, chloral has been found to be so free from disadvantages (and it has been used here to an extent and in such doses as has perhaps not been equalled in any other public institution) that it demands priority over all other drugs in the treatment of the status. But in cases where, on the presumption that some other remedy might produce even more striking results, hyoscyamine has been used it has been found to be almost equal to chloral in the arrest of fast-recurring fits. This was well seen in the instance of a female patient, who had a monthly attack of the status, and in whom chloral stopped the fits for a period exactly proportionate to the dose. Thus half a drachm would ward off the fits for about an hour at a time, and one drachm for about three hours, and so on. In this case hyoscyamine was equally effective in producing the required immunity. In rare cases also, where chloral has been pushed to the greatest extent compatible with safety but unsuccessfully, hyoscyamine has been employed with marvellous results, as will be seen in the following case:—

J. B. was admitted October 24, 1868. He was 24 years old on admission, and appears to have taken fits for 10 years previously. He was almost absolutely demented, could not tell his own name, and was incoherent and fatuous in his conversation. He had 2 or 3 fits daily, and as many in the night, and, over and above these regular seizures, had occasional attacks of the status, in which the fits were severe, and followed each other in rapid

succession. Thirty grains of bromide of potass, given 3 times a day for a whole year, did not diminish the fits either in number or severity. In fact, they became worse, and at times were so severe and frequent that he lost his sight for two or three days at a time. This blindness has since then frequently recurred under the same conditions, and is associated with nystagmus, which prevents ophthalmoscopic examination. It may be added incidentally that another patient, who lived in the same ward, had a similar attack of blindness following upon repeated epileptic seizures. J. B. now took sulphate of atropia for 8 months, but without benefit. The subsequent record of his case is made up of a series of notes regarding attacks of the status, followed by blindness and prolonged stupor, in which he frequently had to be fed with the stomach-tube. Latterly the fits were arrested by repeated drachm-doses of chloral. In the first week of July 1876 he began to have every night 6, 7, or 8 fits, notwithstanding the liberal use of chloral, 30 grains being at first given once or twice a night, and subsequently increased to much larger doses. This, for him, large number of fits was occurring nightly up to July 5, when one-sixteenth of a grain of hyoscyamine three times a day, and a supplementary dose of the same size at 11 P.M. were prescribed. Next night he had one fit, and on the following night none; but this success was again interrupted by the occurrence of 5 fits on the third night after the first administration of the drug. The physiological action had not been induced, and the medicine was ordered night and day, and the full action of the hyoscyamine was developed on July 8. After that date he had only one fit. The physiological action was sustained till the 12th by occasional doses, after which the drug was discontinued. For a period of 35 days and nights he was completely free from fits—an exemption such as he had never experienced or approached to during the whole period of his residence. Both his physical and mental state improved very much, and he could answer questions with more readiness and talk more coherently. His dementia, however, was so deep, that only a slight improvement—that due directly to the absence of fits—could be expected. He gained weight, and was more cheerful. On August 13 he again began to take fits, but at first they were fewer in number than his daily average. During 18 days and nights he had only 17 fits, whereas his common number averaged about 3 per night, the seizures during the day being much less regular and constant. From August 13 onwards he fell into his usual condition, and continues to occupy his place as a confirmed epileptic, for whom nothing but temporary alleviation can be looked for. Still, there can be little doubt that, by inducing at a critical period the physiological actions of hyoscyamine, this patient was delivered from the epileptic status, freed from fits for a period of 35 days, and subsequently for nearly 3 weeks was saved from two-thirds of the seizures which, judging by prolonged observation, he would otherwise have had.

It may not be out of place here to mention the treatment of the excitement of General Paralysis by repeated *large* doses of hyoscyamine given on two or three successive or alternate nights. I have recently had several instances of the

value of the medicines in this condition. The most recent case is that of a General Paralytic who was

Admitted on August 27, 1876. For a whole month he was exceedingly excited and restless, and, notwithstanding the measures employed to subdue him, had only short snatches of sleep. In the intervals he was employed in tossing about, rearranging and tearing his bedclothes, and in smearing the walls and swabbing the floor with his excretions. After 3 repetitions of three-quarter-grain doses of hyoscyamine on alternate nights, his excitement was completely removed. He also became clean in his habits, has since then remained well, and has been transferred to a ward where he may have the privilege of enjoying, for a time at least, such advantages as would have been impossible had his degraded habits remained unchecked.¹

Amongst General Paralytics I have had three or four opportunities of observing a condition which is apt to be troublesome in treatment. Suddenly perhaps the urine which has been passing in a free stream is retained, and no effort can procure its expulsion. This state appears to be due to a spasmodic affection of the *sphincter vesicæ*. A large catheter passes freely till the sphincter is reached, and after a little pressure enters the bladder with a jerk. But repeated catheterisation seems to make matters worse, and rest and soothing suppositories are rather tedious in their action, more especially as mental excitement generally accompanies the retention. In such cases one $\frac{3}{4}$ grain dose of hyoscyamine, followed by repeated $\frac{3}{8}$ or $\frac{1}{4}$ grain doses every three hours (night and day if possible), soon produces marked benefit. A case of this sort was treated quite recently. The patient had had his urine drawn two or three times a day for nearly a week. He was passing through an attack of excitement incidental to general paralysis. On September 29 he had grs. $\frac{3}{4}$ of hyoscyamine. Next morning the medicine having produced sleep, he was much subdued and passed water freely and in a good forcible stream. Small continuous doses were now given, and he went on passing water readily and abundantly. After four days the medicine was stopped, his excitement having abated, and his urination having been established. Next morning the water had again to be drawn,

¹ During the four months which have elapsed since these notes were written the patient has been absolutely free from excitement.

the medicine was resumed, and on the same day he again began to pass water in abundance and with ease. Small doses were given for about twelve days, but the physiological action was never fully established. He could walk about and was free from excitement. The drug was then struck off, the patient being rational and industrious. Since then he has had no difficulty with his urine, and has been subdued and anxious to make himself useful.

One thing is particularly noticeable in the prolonged use of hyoscyamine in suitable cases, and the detection of it removed one of the most formidable objections to the drug. The tongue, mouth, lips, and throat do not become dry, the appetite instead of being impaired improves wonderfully, and mental improvement is marked on the establishment of the physiological action of the drug as shown by the wide dilatation of the pupil, the ataxic pronunciation, and the tottering walk. It is needless to state that similar observations have been made with regard to the prolonged and judicious use of belladonna and opium, and the fact is interesting, as indicating how much the apparently specific actions of a drug are dependent on, or capable of being modified by, variations in the constitutional conditions which the drug has to overcome. One illustration of this and of the value of hyoscyamine in some cases of intense excitement will make these points evident. It was a case which at first was regarded as exceedingly grave, owing to the advanced age, intense agitation and weak physical condition of the patient.

R. T., aged 63, was admitted April 29, 1876. The attack was his first, and it was characterised by symptoms of mania, including violence, delusions, destructiveness, and insomnia. About a week before he was brought to the Asylum, he had, without evident cause, manifested great and sudden violence, and subsequently he, feeling physically ill, threw himself upon his club. After receiving his first week's allowance, he conceived the delusion that he had obtained the money by fraud, and that the police were after him. He became very restless, and wandered about from place to place with the purpose of keeping out of their way. While in the workhouse, during the week previous to admission, he had 3 attacks, in which he became suddenly violent and destructive, and required four men to hold him to prevent him smashing everything in his neighbourhood. In these attacks he seems to have been unconscious, and on one of the occasions asked, on the subsidence of his excitement, if he had had another fit. He was not

convulsed, and his sudden outbreak was not accompanied or preceded by any remark which might have suggested a motive. He had been an abstainer for years, and had no evident delusions of suspicion. In the attacks of excitement he was said to bark like a dog and foam at the mouth; and the barking was subsequently heard in the Asylum. It was said that he had twice tried to cut his throat, but had inflicted no injury.

On admission, 2 ounces of brandy in a bottle of soda water, followed by a draught of chloral hydrate and bromide of potass, were administered. On the first night he slept quietly till 5:30 A.M., when he suddenly jumped wildly from the bed, but did not express any delusion. While in bed in the daytime he again made a sudden spring, and bolted down the passage without evident purpose. Subsequently he showed hallucinations of sight by pointing at figures which, however, he could not describe.

On mental examination he was very taciturn, it being scarcely possible to obtain a single answer from him. He constantly smacked his lips, and emitted moaning, panting sounds, manifesting at the same time much agitation, which, after much pressing, was, he said, due to having 'let them go this way;' but he would or could throw no light on the remark. His tongue was slightly furred. Hyoscyamine in doses of half a grain every 2 hours was ordered on May 1. On the 5th he was much better; he took his food well, and his agitation was much lessened, while the sudden outbursts of excitement had not recurred. He was still very weak, and did not sleep well. The physiological action of the drug had not been established, and his tongue was quite moist. On May 18 there was slight dilatation of the pupils. He was now taking repeated doses of $\frac{1}{8}$ or $\frac{1}{4}$ of a grain according to circumstances. He slept well, and took his food well. At this date the drug was stopped, and next morning all his agitation returned, and the moaning, panting, and smacking of the lips, with other mannerisms which had been gradually disappearing, again showed themselves. The medicine was resumed, and next day he was again as well as he had been before its stoppage. On May 24 a note was taken that he was much better. His mannerisms had gone completely; he talked in a rational and subdued manner, and associated cheerfully with the other patients. His pupils were dilated, but there was no muscular weakness, and his tongue, throat, and lips showed no trace of dryness. This modified physiological action was sustained till June 22. He was then perfectly rational and collected, and as it was considered advisable to send him to his own employment as a tailor, it was first requisite to stop the hyoscyamine, so as to remove the hypermetropia. In the tailors' shop he was very industrious, and frequently expressed his gratitude very cordially. On July 17 he was discharged recovered, having during his period of probation shown no tendency to relapse.

It will be seen that in this case the drug was long in producing its physiological action, and that in fact that action was not fully induced before a recovery was ensured. This leads me to observe that under varying conditions the drug acts with different degrees of rapidity and potency. In

Epilepsy, epileptic excitement, and in violent excitement associated with Chronic Mania, large and frequent doses are required to produce the expected results. In aggressive forms of excitement occurring in Chronic Mania, a quarter of a grain every two hours, night and day, has been given with the production of little effect beyond mental quiescence and dilatation of the pupils. On the other hand, in ataxic muscular conditions and Chorea, usually associated with sclerosis, the smallest doses have been seen to produce most powerful action. In a case of General Chorea which was of nine years' duration, and had resisted every form of treatment in both hospital and general practice, the drug was ordered in doses of $\frac{1}{8}$ th of a grain. By the time of the subsequent medical visit the patient had had 3 doses of $\frac{1}{8}$ th of a grain each. While the medical officer was in the ward, the patient slipped from his seat and fell asleep on the floor. His choraic movements were completely stopped. He was put to bed and soon slept off the effects of the medicine, but the chorea returned. After a period of rest the drug was again given in still smaller quantities. He had $\frac{1}{16}$ of a grain every 3 hours. After the third dose the pupils were dilated, and the movements almost arrested. After the fourth he was still quieter. His appetite was good; he slept well and his tongue was moist, and when roused he talked rationally. The medicine was again discontinued, and for about a week the chorea was not observable. As at the end of that time, however, the twitchings were beginning to return, he was ordered to have $\frac{1}{16}$ th of a grain every night, and the administration of this quantity for about a week running was sufficient to control for a time the involuntary muscular movements. In another case of sclerosis with locomotor-ataxy and great excitement, one-quarter of a grain is always sufficient for the production of mental and muscular quiescence, and half-a-grain causes complete prostration. Such cases contrast very strongly with the instance recorded by me in the fifth volume of these Reports, in which three grains were taken by a healthy man with the production of no other effect than the symptoms of simple mania combined with muscular weakness not amounting to inability to walk and to seize objects,

and followed after six or eight hours by the accession of sleep, interrupted by hallucinations.

There is one condition associated with Mania, and not unfrequently with Senile Dementia with excitement, which is most annoying—the propensity to the tearing of wearing apparel, blankets, &c. When appearing as a symptom of Chronic Mania it is frequently wilful, and is effectually put a stop to by single large doses of hyoscyamine. In one case in the Asylum this mode of treatment is, when occasion requires it, particularly successful. The patient, either in wanton mischief, or in retaliation for some imagined slight or necessary exercise of discipline, either tears blankets or rugs to shreds, or cunningly strips off narrow pieces from their edges, so that for a time his ravages may go on unobserved. Three-quarters of a grain of hyoscyamine invariably put a stop to the intense and aggressive excitement which follows the detection of his wantonness, and the dose keeps him under control for about a month or six weeks, when the memory of his prostration requires to be revived by a repetition of it.

But in cases of extreme Dementia the circumstances are different. The patient may be absolutely unconscious that he is doing wrong, and he demolishes his apparel and bedding in an automatic manner and apparently as the result of a constant impulse. When remonstrated with he can scarcely apprehend the nature of the offence, and when roused to a sense of it, expresses and manifests great contrition; yet as soon as the remonstrance is over the destructive tendency again shows itself. One of the best examples of this variety is at present a resident in the Asylum. He is an old soldier, and shows symptoms of advanced brain-wasting and arterial disease. Night and day his only tendency seems to be to tear. Though very old and feeble, and nearly toothless, he demolishes rugs which appear sufficiently strong to resist the destructive tendencies of a powerful man, and while under observation during the day he attempts at every opportunity to carry his fingers towards the most obscure regions of his apparel in the endeavour to carry out his propensities. These movements are performed in a half automatic half surreptitious manner. Half-drachm

doses of chloral were at first administered to this patient, night and morning, without effect. Every night he destroyed one or more strong rugs. On April 30 he was ordered $\frac{1}{16}$ gr. of hyoscyamine every two hours night and day, and very shortly the physiological action was induced and his tearing was stopped. After the full effect of the medicine was established two days were allowed to intervene, and then he had $\frac{1}{16}$ gr. every night. The good result was kept up as long as the medicine was given, but when it was stopped, and when instead of it 5 gr. doses of chloral were given every three hours, after the action of that drug had been established by a single and much larger dose, he commenced to tear, as the attendant said, 'as ravenously as ever.' When, however, he had had his $\frac{1}{16}$ of a gr. nightly for three months the drug was stopped, and he was not destructive for about three weeks. During the operation of the drug he was able to take exercise in the airing court, and showed little or no muscular weakness; he took his food well, and *his tongue and throat were quite moist*. When in the ward he constantly sat in one position but was not drowsy, and could speak rationally as far as the substance went, but was stammering in utterance and demented in manner. He slept well.

There is one thing with regard to the use of hyoscyamine to which I am compelled to draw attention, though I am unable to explain its occurrence. In this case, when shortly before beginning with the small doses of hyoscyamine, a dose of 1 gr. had been given, the patient had a very severe attack of hæmatemesis. Previous to this another patient to whom a similar dose had been given, had also vomited blood during the period of the action of the medicine: but his case was regarded as accidental, and the vomiting of blood looked upon as a mere coincidence, but on the occurrence of the second case that ground had to be abandoned. In both instances there was a rapid recovery from the effects of the hæmatemesis, and in the one which I have just detailed, small doses were long administered without any return of the vomiting. In looking for an explanation of the phenomenon it was observed, that in both cases there was marked and

extensive atheroma of the arteries, and it was suspected that the changes which the calibre and tone of vessels are felt and seen to undergo during the action of the drug may in these cases have led to rupture of the walls of one or more small vessels.¹ It was consequently determined that large doses of the drug were inadmissible where degeneration of arteries was found to exist. At the same time as the amorphous alkaloid requires a considerable quantity of absolute alcohol and ether for its solution, it was decided to employ 2 ozs. of water instead of 1 in dispensing $\frac{3}{4}$ or 1 gr. of the drug. Since these precautions were taken no case of hæmatemesis has occurred, though the employment of the medicine has been much more extensive since these cases than it was before. I must add that in animals, to which as much as 5 grs. were given hypodermically, no hæmatemesis was ever seen.

There are two forms of mental disease in which hyoscyamine seems to be unavailing. These are Acute Melancholia and Mania with delusions of suspicion in the early, or what might be called the acute form. I have used the drug in several of such cases, but have never seen it productive of decided benefit. I make this statement with the reservation, however, that in the cases in which the drug has been tried other means also have failed, and more extended observation may lead to better results.

There is another class of cases in which hyoscyamine has been used in both repeated small and single large doses with good effect—cases of Recurrent Mania in which during longer or shorter intervals the patients are composed, rational and industrious, but suddenly break out into a form of excitement characterised by great bodily restlessness, a tendency to incessant rapid movement, and an amiable but troublesome officiousness. Experience has taught that a single large dose may cut these attacks short in a night, while repeated small doses exercise little influence on them. Consequently I shall have occasion to refer to them while speaking of the action of single doses.

¹ This view is supported by the fact that in recorded cases of *Hyoscyamus* poisoning, considerable inflammation of the human stomach, with numerous gangrenous spots on the mucous membrane, has been found to exist.

I have frequently observed, and the observation has been confirmed by others, that when small quantities of hyoscyamine are administered at short intervals in cases of subdued excitement, the good effects of the drug are not manifested while it is being used, but on the other hand the excitement superadded by the medicine seems, if anything, to increase the agitation of the patient. Soon after the drug is discontinued, however, a state of quiescence ensues which not unfrequently is permanent. One instance of this sort is recorded in the present volume, amongst the 'Clinical Notes on Conditions incidental to Insanity.' It is the case of C. B.

When in the administration of repeated small doses of hyoscyamine the full physiological action is induced, it generally comes on somewhat suddenly. The patient may continue for weeks showing no symptom except comparative calm, and no sign beyond dilatation of the pupils; when after the administration of the final dose he becomes weak on the limbs, talks incoherently in a husky voice, and becomes the victim of hallucinations of sight. Sometimes he is unexpectedly found on his hands and knees making sudden and ineffectual darts at specks on the carpet, to which he ascribes some illusory importance, and which he cannot reach without repeated efforts on account of his defect of vision.

On passing on to refer to that part of my subject which has to deal with the administration of large single doses of hyoscyamine, I shall first summarise what has been said on that head. In the number of the 'Practitioner' for June 1876, I advanced instances showing the value of the drug in various forms of aggressive excitement, in the excitement of epilepsy, in chronic mania with delusions of suspicion, and in recurrent forms of simple mania. Reference was also made to the care required in administering the drug in acute and senile forms of mania, owing to the obstacle which the dryness of the throat, produced by the drug, placed in the way of the artificial feeding which is so frequently required in such cases. Another caution now requires to be specified in the instance of senile forms of disease, that is, the danger resulting from the use of hyoscyamine where degeneration of the vessels is so apt to exist.

I shall now refer to the employment of the drug in such cases of mania as are characterised by constant and incoherent talking, the treatment of which by this remedy was suggested by the marked suppression of the vocal powers developed during its physiological action. Any substance capable of putting a stop to the logorrhœa prevalent in both the male and female wards of an asylum would be of immense advantage in asylum management. Patients of this talkative class, though usually comparatively innocent in themselves, are so irritating to the violent spirits associated with them, that they constantly provoke assaults and other forms of violence, and sustain a constant current of excitement. It is to be feared that hyoscyamine can exercise at the most only a temporary influence on this form of disease; but even this is something gained. One of the most marked cases of mania of this class was treated in December last by a comparatively large dose of hyoscyamine. The patient was well known as a splendid instance of the possibilities of incoherence. Upon everyone who approached him he poured a perfect torrent of words embodying disjointed and half-expressed ideas, and the number of his digressions and capacity of his vocabulary were limited only by the patience and possibility of escape of his hearers. At the same time he was a good worker, and in every respect well-behaved and obliging. One grain and a half of hyoscyamine (twice as much as would now be given) was administered to him during the afternoon. Three or four hours after he had had the dose, which, strange to say, did not produce marked motor symptoms, he was seen walking restlessly about the bed as if searching for something. He was perfectly silent, and could not be tempted to speak. The next note taken of his case is that shortly after the use of the drug he became much less voluble and more coherent. In addition he expressed a desire to go to work at his own trade, which was that of a tinner. He was at once permitted to do so, and acquitted himself well, though he was still rather loquacious. Since then he has been very industrious, has dined regularly amongst the patients in the hall, never breaks the silence with his talk, and only rarely takes an opportunity

of emitting his outburst of language, which is now much more coherent and controlled. He has never had a repetition of the dose.

In another instance the patient during periods of quiescence was a very industrious man, and reticent, but was liable to recurrent bouts of excitement in which talkativeness was the prominent symptom. One or two of these attacks have been cut short on the first day by three quarters of a grain of hyoscyamine, but the action of the drug in continued doses has not been so effectual. In other similar cases, where about a month of sanity usefully spent in labour has been succeeded by about a fortnight of similar forms of mania, hyoscyamine has been beneficial to the extent of curtailing materially the period of excitement. Lately, however, it has been observed that the drug seems to lose a certain amount of its efficacy when repeatedly given to the same patients. I have not as yet tried the effect of increasing the dose in such cases, but have no doubt that it will prove satisfactory.

In sub-acute forms of mania hyoscyamine has been found exceedingly useful. By subacute cases I mean, in this instance, such as have not run a short, definite, acute course, and still are too recent to be regarded as chronic; and it is frequently found in asylum practice that in many cases that condition has been reached before the patients have been brought in, inasmuch as treatment has frequently been tried unsuccessfully in workhouse insane wards before the patients have been finally certified. I shall adduce only one illustrative instance of the value of the remedy in such cases, as I am certain that it supplies convincing evidence of the efficacy of the drug.

C. L. was admitted in July 1875 with a history of extreme excitement, violence, and incoherence in the expression of exalted ideas. The attack had come on suddenly, and though the patient was said to have been a sober and industrious mechanic, who forsook his special employment to start a beer-shop, it was afterwards discovered that he had been licentious in his behaviour, and that his beer-shop was not above suspicion. On the first night after admission he smashed the door of his single room, and had almost made his escape. He had 45 grains of chloral, and slept for several hours.

All his symptoms pointed to general paralysis, but subsequently he was found to be in all probability a maniac, with delusions of an exalted kind. He was a heavy, powerful man, with a strong stentorian voice, and he employed his physical advantages for the purpose of striking terror into all around him. For a time he was treated with Calabar bean, on the assumption that he was a general paralytic, but no beneficial effect was produced. Chloral also was administered in the course of his frequent outbreaks of wild excitement. During the whole of 1875 his symptoms alternated between quiet but officious good humour and ungovernable outbreaks of excitement. In August it was noted that he was still taking Calabar bean, and was *in statu quo*. In October there was no improvement. He was imperious and self-willed, and had not developed any further symptoms of general paralysis. In December he was very optimistic in his notions, and showed emotional incontinence. On January 5, 1876, a note was made stating that he had been very destructive, had torn several strong rugs, and barricaded himself in a strong single room. He had hallucinations of sight, and imagined that he saw and conversed with a patient who had been dead for a fortnight. On the 3rd he had been intensely excited, and had 1 grain of hyoscyamine, and in a quarter of an hour was asleep. For several hours his sleep was profound, and subsequently he was for some time either in the same condition or, during intervals, was awake but helpless. On the morning of the 5th he was quiet and rational. He remained quieter ever afterwards, and soon requested to be removed from the refractory ward and employed at his own trade as a mechanic. His wish was gratified, and for several months he made himself exceedingly useful, and showed no trace of excitement, and, while laughing at the tentative suggestion of his exalted ideas, he expressed his conviction that he had been very bad, and was willing to work under supervision till his cure was regarded as complete. He was discharged recovered on August 8, and 6 weeks afterwards I was informed that he still continued well and employed himself usefully.

With regard to those cases which were reported in the 'Practitioner' as having been discharged recovered, I have also in some of them obtained information that the patients are doing well. None of them have returned to the Asylum, and none, as far as I know, have had a relapse.

As I have elsewhere entered fully into the consideration of hyoscyamine in the treatment of forms of mania with delusions of suspicion, I shall only refer casually to cases in which the drug has lately been employed in that condition. In one instance, in which the patient was very demonstrative in his expression of delusions that policemen were constantly tracking him, three-quarters of a grain caused next morning complete quiescence. For three days he entirely abandoned his craze, and for eleven days afterwards, though he evidently remained in doubt about his imaginary persecutions,

he could not be got to state that they existed. Similar subsequent administrations have been of equal temporary benefit, and have generally been followed by a desire to go out to work. In another instance, that of a patient who was regarded, in some respects, as the most dangerous man in the Asylum, owing to his constant harbouring of delusions of suspicion, which he expressed only under great irritation, a dose of hyoscyamine was given after the patient had almost succeeded in smashing the head of an attendant with a water-bucket. He slept deeply, and next morning was quieter and more cheerful, and soon expressed himself free from delusions. He was transferred to a quiet ward, and since then has been cheerful, tractable, and industrious, and disowns the fancies which long haunted him. The drug was given three months ago. In the cases which were summarised in the 'Practitioner,' a long exemption from delusions was experienced, and when they have returned the patients have sometimes expressed their conviction of their illusory nature, and asked for the medicine that removed them.

The use of hyoscyamine has been continued in the treatment of recurrent mania with very beneficial results. Instances constantly occur in which the administration of one dose constitutes a turning-point towards sanity. To a patient, for instance, who in his second attack was exceedingly violent and destructive for a fortnight before and three days after admission, three quarters of a grain were given. He had almost succeeded in strangling himself. After the dose he was perfectly quiet and rational, was employed in the kitchen, and was discharged recovered six weeks after his admission, there having been no appearance of insanity after the first four days. It was not an alcoholic case.

Only in one instance, out of perhaps several hundred cases in which the drug has been administered, have I seen a decided rash from the use of hyoscyamine. In many cases there is visible a pinkish, erythematous blush, which sometimes, especially about the face, arranges itself in patches somewhat in keeping with nervous distribution. * But in this solitary instance, the patient, after several doses of

hyoscyamine, was found one morning to be covered from head to foot with an eruption resembling that of measles in arrangement, but darker in colour. The temperature was only slightly, if at all, elevated, inasmuch as it was only 100·8, and the patient had phthisis and laboured under great excitement. Simultaneously with the outbreak of the eruption the excitement was much abated, but it recommenced soon after the stoppage of the medicine. The eruption lasted for ten days, was not associated with or preceded by headache or coryza, and disappeared somewhat suddenly. It was followed by desquamation.

In venturing on a few concluding observations regarding the employment of hyoscyamine in asylum practice, I shall confine myself to a statement of the results of actual experience. It has been gratifying to find that in cases where hyoscyamine has been beneficial in subduing mental excitement, it has not had any tendency such as might have been anticipated, to induce such physical deterioration as takes place when the drug is continuously administered to the lower animals. It is observed that after a single large dose, though the patient may refuse his food during the active operation of the drug, and though the fauces remain dry for about twenty-four hours, it is very rare to see more than one meal rejected; and when the drug is given in suitable cases in continuous small doses, the tongue remains moist to the very last, and the appetite improves *pari passu* with the subsidence of excitement and the addiction to regular sleep.

Another thing is very striking in the employment of the drug, namely, the perfect calm which follows upon its operation—a state of quiescence which cannot be induced by a single dose of any other medicine in ordinary use. When this tranquillity is associated with the re-appearance of all the normal social propensities, and with a desire to resume the exercise of a useful activity, and when, above all, no return of insanity occurs after the direct physiological effects of a single dose have passed off, the value of the drug is placed completely beyond dispute, more especially when, as happened in several of the instances which I have adduced,

excitement in its various forms had been existent for a considerable time before the use of the remedy.

The only other remark I have to make relates to the mode in which hyoscyamine is administered. For reasons already stated, I now use it in a more diluted form than that at first adopted. The formula now employed is

℞ Hyoscyamine, gr. $\frac{3}{4}$.

Sp. Etheris, min. vi.

Alcoholis, min. xvij.

Aq. font. ad. ℥ii.

℥ ut fiat haustus.

The alkaloid is of the amorphous form,¹ and is manufactured by Merck, and supplied by Harvey, Reynolds, & Co., of Leeds. On two occasions I have observed that after standing for two months in a solution according to the formula just given, the drug underwent a great change. It became almost useless, and on holding the solution in bulk so as to view it by reflected light, it was seen to have assumed an olive-green colour, totally different from the tawny brown which is the proper hue of the solution and the alkaloid. On account of this deterioration I have endeavoured, with the kind co-operation of Dr. Major, to introduce the sulphate of hyoscyamine instead of the amorphous alkaloid, but hitherto, on account of the unreasonable price which manufacturers have placed upon the former, I have been unable to do so. Under the circumstances it is necessary to procure only a small quantity of the alkaloid at a time, and to dissolve only such an amount of it as will be sufficient for not more than a month's use. The amorphous alkaloid is comparatively cheap.

Summary.—In conclusion it is necessary to say by way of summary that hyoscyamine appears to me to possess great value in the treatment of cases in which aggressive and destructive excitement is the leading symptom of insanity, in cases of chronic mania with special delusions of suspicion, mania of a subacute or recurrent form, and simple mania, characterised from the first more by agitation than

¹ A description of this extractive principle will be found in the 'Pharmaceutical Journal' for August 23, 1873.

excitement, and due to the existence of obscure delusions and hallucinations. In the treatment of the excitement of general paralysis, in the epileptiform seizures of the same disease, and in the epileptic status it is also of use where chloral, as rarely happens, is found to fail. But perhaps the most striking results from the use of the drug occur in the treatment of such patients as wilfully or impulsively destroy large quantities of clothes and bedding. Prolonged observation has shown how effectually it curbs the wantonness of the one form and soothes the irritability of the other. In wilful destructiveness, three-quarters of a grain or one grain, given in a single dose, reduces the patient to reason, and puts a stop for a considerable period, if not finally, to his expensive habits. In cases where the tearing is carried on as a result of excitement occurring in the course of dementia, a quarter or an eighth of a grain every three hours (sometimes night and day) soon stops the destructiveness and subdues the excitement without inducing bad effects on the general nutrition.

It may be useful, also, to recall shortly some observations which have been made in the body of this paper by stating :—

That hyoscyanine rarely causes a decided exanthematous eruption.

That very rarely (in two cases out of many hundreds of administrations) it causes hæmatæmesis.

That in small continuous doses it does not produce, in suitable cases, dryness of the throat or tongue, and does not interfere with the appetite or induce nocturnal restlessness.

That in cases of retention of urine, occurring in the progress of central nervous diseases, and due evidently to a spasmodic affection of the sphincter of the bladder, hyoscyamine produces free and voluntary diuresis.

That small doses act powerfully in cases of locomotor-ataxy, and other conditions characterised by frequent interrupted nervous discharges, and generally considered to be associated with sclerosis.

That a certain tolerance is established in man, as in the lower animals, by the frequent administration of the drug.

The following cautions may also be useful, viz. :

That in the aged, and in patients showing marked signs of arterial disease, the drug should be administered with great caution.

That it should be freely diluted.

That it should be avoided in cases of furious mania where great excitement exists, and artificial feeding is likely to be required for some time.

That the extractive hyoscyamine should be prepared in small quantities, and kept in a small stoppered bottle to lessen the danger of change from oxidation and other influences.

If these precautions are kept in view, the drug can be extensively employed as a valuable therapeutic agent, and one capable of increasing the recoveries in, and diminishing the expenditure of, lunatic asylums.

THE CLIMACTERIC PERIOD IN RELATION TO INSANITY.

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THE term climacteric has been applied to certain forms of disease occurring, in the male as well as in the female, at the time of life when vigour begins to fail, and the system, having passed the zenith of its development, enters on the first stage of its downward progress. At this period, important tissue changes take place in many of the internal organs, resulting in a greater firmness of structure and probably securing a diminished tendency to variation or derangement of function. In man, as a rule, this season passes with comparatively little notice. The revolution in the system is accomplished gradually and insensibly; there is no abrupt change to mark the different stages of its progress, and the general health is not materially interfered with. In woman, on the other hand, it is always a time of trial, often of suffering and danger. With her the result is more marked, the process is worked out with more general disturbance, and the health is sometimes seriously compromised. 'The immense importance,' says Dr. Tilt, 'of this change on the subsequent life-time of woman cannot be too highly rated, and, as it is well got over or full of suffering, so will the subsequent life-time be healthy or otherwise. It is a *final* settlement; for if it does not develop pathological

seeds fatal to the system, the rest of life is generally passed in uninterrupted health, longevity being more frequently attained by women than by men.'

The phenomena observed at the climacteric age in the female are no doubt in part due to conditions which are common to both sexes. We know that at this period many of the other organs undergo a process somewhat similar to that which takes place in the ovaries after the menopause. For instance, the spleen, the lymphatic glands, and Peyer's patches all suffer a kind of involution. The spleen becomes smaller and of firmer consistence, the lymphatic glands shrivel and waste, and Peyer's patches lose their full rounded form, become more or less flaccid and empty, and at a later age almost entirely disappear. Such changes being common to both sexes at this time of life, may give rise to a class of symptoms, in man as well as in woman, which may be justly termed climacteric.

But the importance of this epoch in the life of the female, as compared with the male, is owing chiefly to the great physiological change which now takes place, through the decay of sexual vigour, and the consequent readjustment of the system to its altered relations in this respect. The generative organs which for a period of thirty years have exercised so powerful an influence, now begin to lose their sway. The ovaries cease to extrude ova, and undergo a process of involution; the menstrual flow, which marked their periodic activity, no longer takes place; and the uterus returns to the state of quiescence in which it existed before puberty. Changes so remarkable stamp this period as one of great physiological importance in the life of woman, and necessitate a readjustment, which is not generally established without more or less disturbance of function in various organs.

The importance of this change, in a pathological point of view, has been variously estimated and some have even denied its influence altogether. The daily experience, however, of those who are most conversant with the treatment of diseases of women, tends to confirm the popular belief in its dangers, and most observers have recognised its

influence as a cause or modifying condition of disease. The frequency and singularity of nervous affections at this period of life are especially noticeable. It is a matter of common observation that the change of life, even when most favourably passed, is accompanied with more or less marked symptoms of nervous disturbance, which, however, generally subside after the complete cessation of menstruation. Dr. Tilt believes some such symptoms to be of almost constant occurrence, and Dr. G. Bedford states that, in addition to structural and malignant disorders so frequent at this period of life, there are many forms of eccentric nervous disturbance, various forms of temporary or permanent paralysis, and the varieties of simple nervous irritation without involving any peculiar lesion are without calculation.

Such being the importance of the climacteric condition in the production of nervous complaints generally, it is only to be expected that it should exercise considerable influence on mental derangement occurring at this period, or even be sufficient of itself to determine an attack of insanity. It is in this relation that I wish to consider it in the following pages.

The results I have to record are derived from an investigation into the cases of patients admitted into the West Riding Asylum during the last four years, and in what I have to say on the subject I shall pursue the following course. First, I shall endeavour, with the material at my disposal, to determine in what proportion of cases the climacteric condition exercised a causative or modifying influence; and, in this connection, I shall notice the influence of the various other causes prevailing at this period of life. In the second place it will be my object to enquire what are the symptoms most frequently characterising mental disease arising in connection with the climacteric change, and to discuss the influence of that change on certain pathological conditions of the nervous system originating independently. I shall then attempt to account for some of the morbid mental phenomena observed at this period; and lastly, I shall indicate what prognosis is warranted in insanity at this period, by the history of the cases investigated.

The great prevalence of insanity, during the period when

the menopause usually takes place, is generally allowed, but the extent to which its occurrence is influenced by the climacteric state has been very variously stated by different observers. For instance, in an article in the first volume of the 'Psychological Journal,' Dr. Reid states that, of 703 cases admitted into Hanwell Asylum during eight years, only 8 were due to the change of life. On the other hand, out of 558 cases admitted into the Edinburgh Asylum during a period of four years, as many as 62 are referred by Dr. Skae to this cause. Dr. Reid's results do not appear to have been derived from personal observation but from the books of the Asylum, and the extreme divergence between his statement and that of Dr. Skae can be attributed only to the imperfection of the records on which his estimate was founded.

The first point that presents itself for consideration in connection with this subject is, whether the age at which the change of life usually appears is more prone to insanity than any other. The following table will furnish some indication as to the relative frequency of mental disease at different periods of life. It shows the ages of 1,054 women admitted into the West Riding Asylum during a period of four years, and the proportion of cases becoming insane during that time to the entire female population living in the district.

TABLE I.

Showing the relative Frequency of Insanity at different Ages in 1,054 Cases, and the Proportion of Insane Cases to entire Female Population.

Age	Number of insane cases	Entire female population	Proportion
Under 15 years . . .	13	211,446	...
15 and under 20 years . .	55	59,960	1 in 1090
20 " 25 " . . .	88	55,659	1 " 633
25 " 30 " . . .	130	50,401	1 " 388
30 " 35 " . . .	135	43,653	1 " 323
35 " 40 " . . .	127	37,311	1 " 294
40 " 45 " . . .	124	33,388	1 " 268
45 " 50 " . . .	111	28,341	1 " 255
50 " 55 " . . .	98	23,982	1 " 244
55 " 60 " . . .	56	17,968	1 " 321
60 " 65 " . . .	49	14,599	1 " 298
65 " 70 " . . .	36	10,217	1 " 284
70 " 80 " . . .	27	10,150	1 " 376
80 " 85 " . . .	5	1,511	1 " 303

From this table it appears that the period of fifteen years from 25 to 40 is the most productive of insanity, the highest number of cases appearing between 30 and 35. Of the entire number 392 became insane from 25 to 40, whereas the period of fifteen years from 40 to 55, which may be considered the climacteric age, furnishes only 333. It is evident, however, that to ascertain the real liability to disease at any period of life we must compare the number of attacks, not only with the number occurring at other ages, but with the number of persons living in the same community at the same age. In this case I have no means of ascertaining the exact numbers of the class from which the patients were drawn, but results sufficiently accurate for comparative purposes may be obtained by taking the entire female population of the districts furnishing patients to this Asylum. The third column of Table I. shows the entire female population of these districts at the different periods of life according to the census returns of 1871. Now if we compare these numbers with the numbers becoming insane we shall find that the tendency to insanity increases with the age up to about the 55th year, after which it suddenly diminishes. Thus while the ratio of cases becoming insane during the period of four years, to the entire female population of the same age, is only 1 in 1,090, from 15 to 20 years, this proportion increases in each quinquennial period at first rapidly, and afterwards more gradually until it reaches a maximum from 50 to 55, being then 1 in 244. In the next period, 55 to 60, it falls to 1 in 320, or about the same proportion as observed between 30 and 35. This diminished ratio is maintained, though with some variation, throughout the later periods of life. Taking the period of 15 years from 40 to 55, the proportion is 1 in 257, while from 25 to 40, the period which yields the highest actual number of cases, it is only 1 in 335, and in the succeeding 15 years, 55 to 70, it is 1 in 303. It appears, therefore, that the period of 15 years from 40 to 55, though it does not furnish the highest actual number of cases, gives the highest ratio to the number of persons of that age living, and is therefore more prone to insanity than any other period of life. How

far this result may be due to causes connected with the climacteric change it is difficult to say, but the sudden diminution in the proportion of attacks after the 55th year would seem to point to the withdrawal of some powerful predisposing or exciting influence about this time, which corresponds with the period of cessation of the menstrual function in a large number of women.

A further indication of the influence of the climacteric condition may be obtained from a comparison of the number of cases occurring in each year with the number of cases ceasing to menstruate in the same years. Table II. shows the number of attacks in each year from 40 to 54 in 333 cases occurring within that period, and side by side with this I have placed Dr. Tilt's table showing the number of cases ceasing to menstruate in each year, out of a total of 436 women in whom the menopause occurred between the 40th and 54th year. A comparison of those two results will show that there is a general correspondence between them, and that the years in which most women cease to menstruate are also those most productive of insanity. Dr. Tilt found that the greatest number of women cease to menstruate in the 45th and 50th years, and it appears from the table the same years also furnish the highest number of cases of insanity.

On enquiring more closely into the history of the 333 cases admitted during this period, it was found that the condition as regards the menstrual function was as follows: In 59, menstruation was regular and showed no sign of failure, and 69 had passed the change some time before the attack, and had enjoyed good health after complete cessation of the menstrual flow; so that in 128 cases there was no reason to consider the change of life as an important element in the causation. In 58 more there was no history of a connection between the mental disease and the menstrual condition, but the records were defective in relation to this point. The remaining 147 were either at the change of life or were suffering from symptoms which had originated in connection therewith.

The following table represents the numbers attacked at

the different ages from 40 to 54, classified according to the condition of the menstrual function at the time.

TABLE II.

Showing Age at the time of attack, and the condition of Menstruation in 333 Cases.

Age	Menstruation regular	At change, or with symptoms referable to it	Past change	Not classified	Total	Number ceasing to menstruate in 443 cases (Dr. Tilt)
40 years .	18	10	0	4	32	42
41 " .	12	5	0	4	21	17
42 " .	10	13	2	3	28	26
43 " .	5	8	2	3	18	24
44 " .	8	13	2	2	25	23
45 " .	2	17	2	5	26	49
46 " .	1	14	3	1	19	31
47 " .	1	12	1	1	15	42
48 " .	1	12	8	7	28	37
49 " .	1	7	8	7	23	32
50 " .	0	20	9	7	36	49
51 " .	0	6	8	1	15	27
52 " .	0	4	9	4	17	16
53 " .	0	3	5	3	11	9
54 " .	0	3	10	6	19	7
Total .	59	147	69	58	333	443

Hence it appears that the age most liable to an attack of insanity in connection with the change of life, is the period from the 44th to the 48th year, which, it will be seen, furnishes as many as 68 out of 147 cases. Though the 50th year shows a higher number than any other single year, yet the five years subsequent to the 48th furnish only 40 cases as compared with 68 in the period of five years preceding. This decrease is greater than the ratio of decrease for the corresponding period in the number of cases ceasing to menstruate as shown in Dr. Tilt's table, a fact which, so far as it goes, indicates that the liability to the occurrence of insanity at the menopause decreases as the age advances. The numbers, however, in both cases are too small to furnish reliable results. The average age of the entire number of cases occurring between 40 and 54 was 46·5.

Besides the 147 cases occurring between 40 and 54, there were 12 cases at ages under 40 or over 54 in which the

attack was referred to the menopause. Of these 2 occurred at 55, and 2 at 57, 3 at 39, 4 at 38, and 1 at 36. Thus out of 1,054 cases admitted during four years, 159 in all were at the change of life or had symptoms referable to that cause.

It by no means follows, however, that in all these cases the climacteric condition was the only or even the chief element in the causation of the mental disorder, though it may be affirmed that in most cases it exercised a causative or modifying influence more or less marked. The history of the cases investigated points to the conclusion that the change of life is not often of itself the immediate cause of insanity. The outbreak of the disease is generally determined by some other influence most frequently of psychical character, but sometimes also by bodily disease not connected specially with the climacteric age.

In 76 cases out of 149, a history of some such exciting cause could be made out, and the following were the influences chiefly at work :—

Death of relatives in 15 cases ; domestic trouble, chiefly connected with cruelty or drunkenness of the husband, in 18 ; pecuniary difficulties, in 5 ; fright, in 6 ; disappointment in love, 2 ; remorse, in 3 ; ill health and want, in 4 ; menorrhagia, in 4 ; fall or injury to the head, in 3 ; fevers and inflammations, in 4 ; intemperance, in 5 ; disease of the heart, in 3, and phthisis, in 4. In addition to this, almost all cases had a history of derangement of the general health for some time before the attack, although not suffering from any special complaint.

In regard to alcoholic intemperance, it may be here remarked that in 11 of the 147 cases there was a history of intemperate habits ; but in 6 of these the craving for alcohol had shown itself first at the change of life, and is, therefore, to be looked upon rather as a morbid symptom than as an exciting cause. In the remaining 5, the intemperate habits were of long standing, and formed doubtless an important element in the causation. The influence of alcoholic intemperance is more felt in women indirectly through drunkenness on the husband's part and consequent anxiety and worry to the wife.

In 31 of the cases there existed various forms of organic disease of the brain, which do not appear to have been, in most cases at least, specially connected with the climacteric condition. Of these 11 were suffering from epilepsy, 5 from general paralysis of the insane, 6 from embolism or cerebral hæmorrhage, and 9 from atrophy and other forms of brain disease.

In addition to the various influences, moral and physical, already mentioned as exciting causes, there are certain conditions, in addition to the climacteric state which may exercise a special predisposing influence at this period of life. Under this head I shall remark on the influence of marriage and childbearing, hereditary predisposition and former attacks.

The condition of a patient as to marriage might readily be supposed to exercise some influence on the tendency to mental disease at this period. There is a popular idea that married women get over the change of life with less trouble than single, but this opinion does not appear to be in accordance with the experience of those most capable of judging. As regards insanity at this period, the facts brought out by a comparison of the numbers of married and single women attacked, seems at first sight to indicate that the latter are much more liable to mental disease at this period. Of 147 women becoming insane at the change of life, 90 were married, 33 single, and 24 widows, in other words, of every 100 women becoming insane, 61·2 were married, 22·5 single, and 16·3 widows. Now, in the entire female population of the West Riding of Yorkshire, of ages varying from 40 to 54, the percentage of married, single, and widowed persons is, according to the census returns of 1871, 76·57 married, 10·07 single, and 13·36 widows. It thus appears that the percentage of single women becoming insane at the change of life is more than double that which exists in the general population at the same age, while the proportion of married women affected is less. If, however, we take the cases becoming insane at the corresponding period of life from causes independent of the change of life, we find the proportions of married, single, and widows to be, respectively, 117, 38, and 31, or almost identical with the proportions found in the

cases connected with the change of life. Hence it would seem that, though single women are undoubtedly more liable to insanity at this age than married, the result is not attributable to causes connected with the menopause, but to other influences incidental to this term of life. It is probably due in some measure to the helpless and unprotected state of unmarried women at this age.

With regard to the influence of childbearing, the cases examined afford no evidence that the liability to insanity is in any way affected by the number of children a woman may have borne. Attacks appear to be as frequent in those who have had no children or only small families as in those who have had many.

Hereditary predisposition was found to exist in 43 cases, or about 28 per cent. In a good many cases, however, no family history was obtained, and in still more it was only imperfectly ascertained. It is probable, therefore, that the proportion of cases hereditarily predisposed was much larger than would appear from the records.

Former attacks had occurred in 49, or exactly one-third of the entire number of cases,—a fact which shows that the change of life is specially apt to cause an attack, if the patient has previously been insane. In the recurrent cases the periods of life when the first attacks occurred were, in 13 cases, at ages over 40; in 29 cases between 30 and 40; and in 7 cases between 20 and 30. In 13 of the recurrent cases the attacks had been puerperal or connected with lactation.

Having noticed the various influences in addition to those connected with the change of life tending to produce insanity at this period, we come now to enquire whether there is any class of symptoms whereby we can recognise cases of mental derangement due to the climacteric condition.

In the production of insanity, as of other diseases, it is not usually to any single specific cause that the resulting morbid phenomena can be attributed, but to a combination of causes acting together, and each making its influence felt more or less powerfully according to circumstances.

When we consider, moreover, that the constitutions affected vary physically and mentally as the extraneous influences affecting them, it is not surprising that the effect of any particular causative agency should be very variously expressed in different cases, or that it should be altogether impossible to appreciate the effect due to each circumstance in the chain of causation. But if there is present a condition exercising so powerful an influence on the system as the climacteric state confessedly does, it is reasonable to expect that the symptoms of mental disease originating under such circumstances should present a certain amount of uniformity.

The late Dr. Skae gives, as pathognomonic of the insanity of the climacteric years, a group of symptoms characterised by 'a monomania of fear, despondency, remorse, hopelessness passing occasionally into dementia;' and Dr. Maudsley, speaking of the mental derangements of this period, observes: 'When positive insanity breaks out, it usually has the form of profound melancholia, with vague delusions of an extreme character, as that the world is in flames, that it is turned upside down, that everything is changed, or that some very dreadful but undefined calamity has happened or is about to happen. The countenance has the expression of vague terror and apprehension. In some cases short and transient paroxysms of excitement break the melancholy gloom. These usually occur at the menstrual period and may continue to do so for some time after the function has ceased.'

Such a condition is no doubt very common in connection with insanity at the change of life, and similar symptoms were observed in a large proportion of the cases originating at this period, whose history was investigated. But there seems to me to be another class of symptoms almost equally common, consisting in the early stages of the disease, of perversion of the affections, distrust and vague suspicions of relatives and friends, and developing at a later stage into well marked delusions of suspicion and persecution. Both these groups of symptoms seem to be highly characteristic of insanity connected with the climacteric condition, but I do

not think that an observer, unacquainted with the age and history of the patient, would be warranted in pronouncing a case presenting such a group of symptoms to be one of climacteric insanity. To be of diagnostic value the symptoms must be taken in connection with the early history and development of the disease, which in most cases observe a pretty uniform course. Before any attention is called to the mental condition, the patient has, in the great majority of cases, been for some time more or less out of health, anæmia, and debility in some form, being generally present. Probably she is reduced by previous severe menorrhagia, although the occurrence of this condition in connection with the menopause does not seem to predispose specially to an attack of insanity. In other cases, though menstruation has not entirely ceased, it has been for some months scanty, irregular, and painful. In most cases, however, the menstrual flow has entirely ceased some time before mental symptoms are developed, but in the interval the patient has never been restored to her former good health. She continues to suffer from various anomalous sensations, faintness, and sinking, headache, palpitations, epigastric pains, and feelings of general debility, with flushings and perspirations. At this time there often appears a craving for stimulants, and not unfrequently habits of intemperance become developed. So far the condition of the patient is one common enough in women at this period, and occasions no remark on the part of others. Even at this stage, however, the patient herself is often conscious of some mental defect, forgetting where she puts things, and not being able to get on with her work as before. She may have a vague feeling of something being wrong, though not sufficiently intense to prompt her to do anything obnoxious to others or injurious to herself. At the same time she is irritable and restless, anxious about trifles, and unusually susceptible to the influence of all stimuli. Occasionally she experiences a strong impulse to certain acts often of a suicidal or homicidal nature. In one woman this impulse took the form of a desire to kill some one near and dear to her, and she confessed that, before anyone noticed her mental condition, she experienced this

feeling, and had to leave the house of a daughter with whom she was living, on account of an uncontrollable impulse to murder her grandchildren.

In some cases depression is an early symptom, and the patient may confess that she feels tired of her life, and has entertained thoughts of suicide. More generally, however, attention is first called to her mental condition by some marked change in her character and conduct, such as neglect of her household duties, or an amount of fussiness in the performance of them quite foreign to her usual disposition. In one case, for instance, a woman noted for regularity in her habits and economy in the management of her household affairs, took to getting up at irregular hours in the night for the ostensible purpose of getting through her domestic duties, with which, however, she never made any progress. The same patient was in the habit of lighting large fires in every room in the house with no apparent object, and would spend a great part of her time in making meaningless rearrangements of furniture. Another whose former life had been marked by devotion to the interests of her husband and family, and whose delight was found in making home pleasant to them, became neglectful of home, and spent most of her time in going about from house to house collecting and retailing all the gossip of the neighbourhood. With such changes in the habits and character there is always depression more or less marked, and often an inclination to suicide. The patient manifests great general restlessness, extreme nervous irritability, and, it may be, distrust and vague suspicion of those about her. She has a sense of dissatisfaction with everything she does, and, though always busy, can never get through with her work. A very constant symptom at this stage is a tendency to wander away from home without any expressed object on the patient's part, but probably under the influence of a vague sense of distrust and suspicion which has not yet assumed any definite shape.

Such is in general the early history of insanity, originating in connection with the change of life; but as the disease progresses, different cases may assume different forms according to circumstances and surroundings. Most

frequently the depression deepens into profound melancholia marked by a condition of fearfulness and apprehension of some impending calamity, with vague delusions of a gloomy character. In other cases the distrust and suspicion of others, manifested at an earlier period, become the most prominent symptoms, and develop into well-marked delusions of persecution, under the influence of which the patient may become violent and abusive.

Looking to the symptoms displayed at the time of admission, by the cases whose history was investigated, it seems to me that, excluding cases complicated with organic disease of the brain, they might be all referred to three classes, characterised by the prominence of different groups of symptoms.

The first class was characterised by simple depression without hallucinations of the senses or intellectual derangement. In some cases there was extreme nervous irritability and hyperæsthesia of sensation almost amounting to hallucination.

In the second class also, depression was the prevailing mental condition, but along with this there was great emotional and intellectual disturbance. Hallucinations of the senses were not uncommon, and some vague delusions of a depressing kind were nearly always present.

The third class of cases presented delusions of suspicion and persecution as the most prominent symptom. In most cases hallucinations of the senses were present, and outbursts of excitement not unusual.

But in addition to cases of uncomplicated insanity we have seen that among those investigated there were several suffering from various forms of brain disease not specially connected with the change of life. These present for the most part symptoms peculiar to themselves, but it may not be without interest to enquire how far, if at all, those symptoms were modified by the climacteric condition. I shall notice successively the cases of epilepsy, general paralysis, and atrophy, and other forms of brain disease.

The history of the cases admitted suffering from epilepsy at the change of life shows that the climacteric condition

may not only exercise an important influence on the symptoms of this disease already existing, but may in some cases be the originating cause of it. In one case at least the disease occurred for the first time at this period, and could be referred to no other cause.

C. M., a married woman, aged 48, enjoyed good health till her 45th year, when menstruation ceased. She immediately after became peevish, quarrelsome, and fretful, and manifested groundless distrust and suspicion of her friends. Epileptic fits came on about the same time, and recurred almost every month for the next two years. After that they took place at longer intervals, and have been getting gradually less severe. She was brought to the Asylum in consequence of having tried to strangle herself in the excitement following an attack of fits. The fact of the monthly occurrence of the fits in this case leaves little room for doubt that the fits are due to the periodic return of the menstrual nixus, and the fact that they are becoming more rare and less severe as age advances, raises the hope that, when the system has fully adapted itself to its altered sexual relations, the seizures may entirely cease.

In the case of 9 other epileptics admitted at the change of life, the symptoms had become aggravated. Some of these present points of interest, and deserve more particular notice. In one case the fits came on at puberty and continued; but gradually diminishing in frequency and severity till 20 years of age, when they ceased entirely. Menstruation continued regular through life, and there was no return of the fits till the menstrual function began to fail, when the fits again reappeared. In another case epilepsy came on first at puberty without any other assignable cause, and continued to occur occasionally at the menstrual periods, up to the age of 23, when the patient married. She had several children, and seldom suffered from fits during her married life except after confinements. At 42, menstruation began to fail, and from that time her condition became aggravated. The fits, which had formerly occurred without much mental disturbance, were now accompanied with great excitement, which at last became continuous, though the fits took place only at the monthly periods. Menstruation entirely ceased after about a year, but no improvement took place in the mental condition. She got rapidly demented and still remains in that condition. The fits continue to

recur about once a month, and are accompanied with considerable excitement.

The other cases presented nothing worthy of remark; most of them had suffered from epilepsy for many years, but had not been regarded as insane till the cessation of menstruation brought on excitement. With regard to general paralysis, only 5 cases were at the change of life on admission; and it is worthy of note that in all these the symptoms were of a melancholic type, suicidal tendencies having been very pronounced in two of the cases. One, on admission, was suffering from symptoms of simple melancholia, and a favourable prognosis was entertained of her case. She had no delusions of a grandiose character, and none of the usual physical signs of general paralysis. She was quite rational and intelligent on most subjects, but confessed she had a desire to commit suicide, and had thought of all ways of accomplishing her object. The reason she assigned for this was a feeling of intense misery; in her own words, 'She did not know what to do with herself, or where to put herself.' The only physical symptom noted on admission that seems to point to general paralysis was a complaint of 'cramps and twitchings' in her legs, which she herself attributed to rheumatism. She continued unchanged for the first month after admission, and during the next six months the condition of depression alternated with fits of excitement, which became more and more frequent till at the end of a year well-marked symptoms of general paralysis were developed. From that time the condition rapidly deteriorated and the disease pursued its usual course towards a fatal termination the symptoms being, however, of a melancholic type throughout.

The history of this and the other cases indicates that the climacteric condition is capable of exercising a modifying influence of a depressing character on the symptoms of general paralysis arising at this time, and I believe the same condition will be found present in most patients suffering from this disease while passing through the menopause. There is, however, no reason to suppose that general paralysis is specially apt to originate at this time, or that the

relation between the two conditions is other than accidental.

The same remarks apply to the cases of apoplexy, atrophy, and other organic diseases of the brain, all of which are found to occur at the menopause, but, in the great majority of cases, owing to causes incidental to this time of life, and entirely independent of influences directly connected with the change of life.

The liability to mental disease at the change of life seems to depend on the peculiar susceptibility of the nervous system which exists at this period, as evidenced by the manner in which it responds to the stimulus emanating from the reproductive organs and to other influences. This condition manifests itself more or less strongly at all periods of life in connection with ovarian activity, with which it seems to be specially connected. Even at the ordinary menstrual periods the susceptibility of the nervous system to all kinds of stimuli is much increased, and many of those modifications of the temper and feelings, which in other circumstances become permanent conditions, may then occur in an evanescent form. It is a matter of daily observation that at those periods most women are irritable and capricious and more susceptible to the influence of cold and other physical agents as well as to emotional stimuli; while some exhibit a disturbance of the nervous system almost amounting to disease. The same condition is exemplified in the nervous derangements connected with the establishment of the menstrual function, and in the well-known mental peculiarities developed during pregnancy and the puerperal condition. But it is more especially at the climacteric period that this sensitiveness of the nervous system is manifested.

In searching for an explanation of the phenomena observed in mental disease at this period, we have, therefore, in the first place, to enquire into the origin of this condition of nervous susceptibility, and then to show how it conduces to the development of the more pronounced symptoms of insanity.

Without assuming any theory with regard to the nature of nervous energy, it may be asserted that this condition of

affectability, which forms the groundwork and starting-point of mental derangement, is due proximately to some alteration in the constitution of the nerve tissue, in virtue of which it becomes less stable and discharges its energy under the influence of stimuli that would not in other circumstances suffice to call forth its peculiar activity. Now there are only two ways in which such a change in the constitution of any organ can be brought about, namely a change in the nutritive material supplied, or an alteration in nutritive activity of the part itself. The condition of nervous irritability arising in connection with ovarian changes at the climacteric period must, therefore, depend on some change in the blood supply, or on altered nutrition in the nervous centres arising spontaneously, or induced by influences emanating from the reproductive organs.

The blood may affect the nutrition of the brain, either by an increase or diminution in the supply, or by an alteration in the quality. It will not be difficult to show that the condition under consideration is not the result of cerebral hyperæmia; for it is found much more frequently in weak and anæmic than in plethoric subjects. And the same may be said in regard to cases of actual insanity; for in those who become insane at the change of life the attack is much oftener accompanied with a deficiency in the amount of blood than with the opposite condition. Nor will the condition of anæmia, on the other hand, account for the production of nervous symptoms in all cases, although it may be an important factor in many. It is a well-known fact that want of blood from sudden hæmorrhages is a common cause of fainting, convulsions, and other nervous phenomena; and when cerebral derangements arise in connection with menorrhagia at the change of life they may doubtless sometimes be explained in the same way as the phenomena due to too copious bleeding—by the withdrawal of the stimulus of the healthy blood-supply from the brain. In the great majority of cases, however, there is no such history, and the symptoms of nervous irritability are often displayed when there is no evidence of anæmia, and the tissues present every appearance of healthy nutrition.

With regard to the influence of a change in the quality of the nutritive material, it has been supposed by some that this condition of nervous susceptibility is due to poisoning of the blood by the retention of something which should have been eliminated by the menstrual flow. This theory would not explain the occurrence of the phenomena observed at the ordinary menstrual period, for if the irritable condition of the nervous system then observed were due to poisoning of the blood, it would be manifested not so much during ovarian activity as before the occurrence of the menstrual flow. Again, it has been observed by Dr. Jebb and others that hysteria and other affections evidently referable to ovarian influences occur in young girls long previous to the first menstruation, showing that even at an age when there is no idea of sex, the influence of ovarian evolution may be apparent in producing an irritable condition of the nervous system. It appears, therefore, that the condition cannot be attributed to poisoning of the blood any more than to cerebral anæmia or hyperæmia.

But if the change in the quality of nerve tissue is not due to an alteration in the quantity or nature of the nutrient fluid, it must depend upon some alteration in the nutritive activity of the tissue itself. Now such a change may either originate spontaneously in the central nervous system, or be induced by some influence emanating from the reproductive organs. We may suppose that, in virtue of the original endowment of the nervous system, there occurs, at the change of life, a spontaneous alteration in the nutrition of the centre presiding over the sexual function, correlative and antecedent to the changes which take place in the ovaries and other organs at this period. And no doubt something of this kind takes place, but it is equally certain that ovarian changes, induced by the influence of the centre directing the activity of the sexual system, are capable of exercising an important reflex influence on the cerebral functions through the medium of the nervous supply. The genital organs are abundantly supplied with sympathetic nerves, and through these have intimate connections with the various ganglionic centres, as well as with the cerebro-spinal system. The

common occurrence of epigastric faintness and sinking, and other anomalous sensations in women undergoing the change of life, as well as in other states of ovarian activity, shows the intimate relation that exists between some of the sympathetic centres and the reproductive organs. The changes taking place in these organs, then, seem to react in the first instance on the abdominal ganglionic centres, causing them in their turn to influence the brain; and it would appear to be in this way chiefly that the nervous susceptibility which we have seen to exist in connection with ovarian activity is produced. It may be that the sympathetic system exercises over the nutrition of the brain some influence similar to the control it is acknowledged to have over the bloodvessels and secreting organs. If so, the reflected irritation of ovarian changes would tend to disturb this influence, and so cause an alteration in the nutrition of the brain, resulting in the modified tone of nerve tissue which shows itself in nervous affectability.

Now it is easy to understand that such a modification in the tone of the nerve tissue should form a favourable soil for the development and growth of more decided mental symptoms. We know that there is the most intimate connection, the most perfect sympathy, between the various organs in the body. No change takes place in any part but has its correlative condition in the central nervous system. The brain contains in itself the organic expression of the condition of every part, and is, so to speak, organically aware of every change that takes place whether in structure or function. In ordinary circumstances, however, this connection is not a subject of consciousness, and, in a healthy condition of nerve tissue, even considerable deviation from the normal state of any organ may take place without the condition becoming consciously known, although organically expressed, in the brain. If, however, there exists such a modification of nerve structure as to render it more affectable, the brain will necessarily be much more sensitive to the influences emanating from the various organs. The slightest derangements in the functions of any organ, or even ordinary physiological processes, may then have a tendency to force them-

selves into consciousness, manifesting themselves in the sensory centres by pain or some abnormal feeling, and if the condition continues and reaches a certain intensity it may show itself in the higher centres by perversion of the emotions and the will, and in actual delusions.

The development and growth of emotional perversion and delusions from the condition of nervous susceptibility existing at the change of life will be best shown by a short record of one or two cases. The first that I shall refer to is one in which extreme nervous sensibility was the chief symptom of the disease, though there were occasional attacks of intense depression.

J. S., a married woman, the mother of two children, the one 25 and the other 9 years of age, enjoyed good general health till her 42nd year. At that time the menstrual flow began to fail, and after continuing scanty and irregular for another year, ceased altogether. During the 'dodging time' she suffered from epigastric sinking, occasional pain and lightness in the head, and various anomalous sensations about the chest which she described as 'spasms and tight breathing.' On the entire cessation of menstruation these abnormal sensations, instead of disappearing, became more pronounced, and she now began to suffer from a distressing feeling of 'emptiness' in the crown of the head and great hyperæsthesia of the senses. 'My feelings,' she herself said—meaning sensations—'were complete torture to me.' At the same time she was extremely susceptible to the influence of emotional stimuli, and the sight of anything painful or disagreeable caused her the greatest anguish. A few weeks after this she experienced one morning a feeling of the most intense unhappiness, which passed off in the course of the day, and did not appear again for some months, when attacks of depression returned, and became more and more frequent. This condition continued, now better, now worse, till about two years after cessation of menstruation, when the depression became so intense that she felt tempted to commit suicide, and requested her friends to have her taken to an asylum. The symptoms on admission were such as have been described, and her condition during her residence in the asylum was characterised by extreme sensitiveness and attacks of intense depression without intellectual disturbance. Under tonics and mild sedative treatment she improved considerably while in the Asylum, but was removed by her friends before the cure was complete.

The above case shows an extreme degree of that nervous irritability which is always more or less present at the change of life, and it will be observed that, though the depression was sometimes so great that she contemplated suicide, she was never the victim of hallucinations or delusions. There

is little doubt, however, that, had the condition continued, the morbid sensations would have reacted on the higher nervous centres, so as to produce actual delusions.

The following case illustrates the production of actual hallucinations and delusions in connection with similar anomalous sensations arising at the cessation of menstruation.

E. S., aged 52, the mother of twelve children, the youngest of whom was 9 years of age, enjoyed uninterrupted good health till her 50th year, when the menstrual flow, after dodging her for a few months, ceased. From that time she suffered from periodic headaches and various abnormal feelings in different parts, the most notable of which were a feeling of heat at the pit of the stomach and a constant pain in the left side. All this time she manifested an amount of peevishness and irritability not natural to her, and a great dissatisfaction with everything about her. No distinct mental derangement showed itself, however, till about two years later, when she began to manifest suspicion of her friends and neighbours, and to suffer from hallucinations of hearing, under the influence of which she became excited, and had to be brought to the Asylum. On admission she was somewhat agitated, looked frightened and suspicious of everybody, and, when questioned, stated that she constantly heard voices calling her all sorts of opprobrious names. In these voices she recognised some of her neighbours, whom she accordingly imagined to be concealed about there. She had the delusion that they had persecuted her, and told lies about her, also that they had invented a machine for projecting invisible darts through the air, by means of which they could at any time torture her. She could give no explanation of the nature of these darts, but said that they entered her body about the region of the chest and stomach, producing a burning sensation like scalding. She indicated, as the part upon which they mostly played, that region of the left side where she had suffered from pain previous to the attack.

In this case, as in the last, we have a slight alteration in the organic condition of certain parts, forcing itself into consciousness as an anomalous sensation, but here the sensory effect reacting on the supreme nervous centres, induces in them a corresponding change, resulting in the growth of a morbid idea correlated to the morbid organic sensation, and manifesting itself in delusions.

Numerous cases might be given illustrative of the development of delusions in sympathy with morbid organic sensations arising from ovarian influence, but the above will suffice, and I must now pass on to consider the last subject

I proposed at the outset to discuss, namely, the prognosis to be expected in insanity at the change of life.

The prognosis in insanity occurring in the climacteric years is stated by many writers to be unfavourable as regards recovery. Drs. Bucknill and Tuke in their 'Manual of Psychological Medicine,' express this opinion, and Van der Kolk, speaking of this subject, says: 'If religious melancholia begin in the climacteric years, then the prognosis is very unfavourable; generally incurable mischief of the uterus occurs; the plethora of the uterus and the reflex symptoms depending upon it continue, and make the disease incurable.' The history of the cases which I have investigated, however, shows that as regards ultimate recovery the prognosis is by no means unfavourable, though an early recovery is not generally to be expected. Of 333 cases in whom insanity occurred between the 40th and 54th year, 149 or 44·7 per cent. recovered. Of the 147 of these in whom the attack was referable to the change of life, 69 recovered, or nearly 47 per cent., so that the recoveries were somewhat more numerous in those that were at the change of life than in those whose disease could not be connected with this cause. If we exclude the cases complicated with epilepsy, general paralysis, and other diseases of the brain, the proportion of recoveries is as high as 59·5 per cent.—a very high proportion of recoveries. The prognosis may, therefore, be considered very favourable in uncomplicated cases.

CASES ON THE BORDERLAND OF INSANITY.

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A DEFINITION of Insanity, logically speaking, is an impossibility. Medicine, not being an exact science, we can do no more than give an approximate description of the various diseases which fall under our observation. Nevertheless, there is no occasion for psychologists to throw a veil of mystery around the term Insanity itself, nor around those subdivisions of it which appear so paradoxical to the legal profession and to the public at large.

Perhaps no term has been more freely used or more variously applied than that by which we designate the condition of those who are not quite insane—the Borderland of Insanity.

First let us endeavour to discover what it is not.

Setting aside physical complications, such as epilepsy, chorea, and paralysis, and confining ourselves for the present simply to mental symptoms, it is contended that pure hysteria, pure hypochondriasis, and pure eccentricity are conditions which are as much outside the Borderland of Insanity, as mania, melancholia, and dementia are within it.

When, on the contrary, hysteria becomes complicated with mental symptoms, of however slight a character, it

appears that it ought undoubtedly to be considered to be a disease on the Borderland of Insanity.

We would therefore be understood to recognise three separate conditions.

The first includes such disorders as hysteria, hypochondriasis, eccentricity, etc.

Secondly. The Borderland, separated by what may be conveniently called,

Thirdly, the Boundary Line from Insanity.

In order that the object of this paper may be understood, an attempt must be made to define, however roughly, that condition known as the Borderland of Insanity.

For the present purpose it may be allowed that patients on the Borderland of Insanity are those who are suffering from distinct mental symptoms, which are yet not so marked as to justify a medical man in signing a certificate stating that they are insane.

Those, on the contrary, whose mental symptoms are so decided as to justify a practitioner in signing such a certificate, are, for the present, to be regarded as patients who have crossed the boundary line separating sanity from insanity.

When we closely investigate these Borderland cases, we find that they exist in certain natural groups.

The writer divides them in the present instance into five groups, which are not to be considered as necessarily exhausting the subject, but as rather including under convenient subdivisions the cases most commonly met with in private practice.

The first group includes two cases of moral depravity.

The second, two cases complicated with hysteria.

The third, two cases of congenital imbecility.

The fourth, two cases with premonitory symptoms.

And the fifth, two cases of convalescence from insanity.

Two cases are reported in each group. The first case in each group is one on the Borderland of Insanity. The second represents a patient who has overstepped the boundary line and become actually insane, although in these latter cases the mental symptoms are very faintly marked.

I.

With regard to the first group—Moral Depravity—we would venture to make a few observations.

Dr. Prichard has stated in his 'Treatise on Insanity,' published in 1835, that 'insanity exists sometimes with an apparently unimpaired state of the intellectual faculties.' With all deference to this great author, we may remark that we do not believe that such a condition ever existed, except on paper. The writer is frequently consulted by the parents of children, both in the upper and lower classes, who are at their wits' ends to know what to do with a son or a daughter whose 'naughtiness' is beyond all control. After a careful examination of several patients suffering from the so-called 'Moral Insanity' of Dr. Prichard, the writer can confidently state that he never yet found a case of this description which was not more or less complicated with Imbecility. Cases of moral insanity are either cases of moral depravity or of imbecility, or of the two conditions combined.

It is in the upper classes that the larger proportion of such patients are brought under the notice of the medical adviser. The parents are more in a position to pay others to keep these troublesome individuals out of mischief, and an *exposé*, stamping the family with a suspicion of insanity, is more dreaded by them than it is by their poorer brethren. Amongst the lower orders, such cases go either at once to prison, being less carefully watched, or first to a reformatory and afterwards to prison, a very small proportion finding their way into public asylums.

First Group.

Case 1.—Moral depravity. Mental symptoms not so marked as to justify a medical man in signing a certificate of insanity.

Miss B., æt. 19; the daughter of a captain in the army; a tall, robust-looking girl, of a lively temperament. When a few months old had an attack of meningitis. She has always been wilful and troublesome as a child. About a year ago a strange set of symptoms were developed. The patient took off some of her clothes, stuffed them up the chimney, and set

fire to them. Upon the servants rushing in to extinguish the flames, they found her sitting on the hearth-rug, clapping her hands, and saying, 'What a fine blaze!' She had on several occasions destroyed clothing, furniture, and books. Her methods of destruction were most ingenious. She would carefully cut all the string which bound the pages of a book together, and then leave it on a table, so that if any unsuspecting visitor handled it, it would fall to pieces. On one occasion she drenched a baby, who was sleeping in its cot, with water, and she was in the habit of deluging her room with water, for no reason whatever.

She was decidedly backward for her age, although her education had not been neglected. She could read and write, but could not keep accounts. She preferred to read juvenile books, such as are only suitable for mere children, she being 19 years old. The catamenia were regular, but the discharge was somewhat excessive. The patient had attempted to throttle her attendant, and had also stated that she thought suicide was a commendable act. There was a history of self-abuse. She was perfectly coherent and rational in conversation. She accused others of having done the mischievous acts attributed to her, but acknowledged that she had often contemplated doing them when alone in her room.

The patient had a propensity for falling in love with every man she saw, and as the physician with whom she had resided also received male patients, her removal from his house became necessary. No medical treatment by drugs was desirable in this case. The question to be solved was—What was to be done with her? It was evidently not a case for an asylum, nor for home treatment. The patient was therefore sent to a clergyman's house, from whence, after a residence of some months, she was discharged recovered.

Case 2.—Moral depravity. Mental symptoms sufficiently marked to justify detention in an asylum.

Miss L. E., æt. 29; admitted to my establishment, under the care of Dr. Blandford, October 30, 1874; single; fourth attack. This was an extremely difficult case to certify. No delusions could be detected. The patient simply made herself objectionable to her friends, but in such a manner as to leave no doubt in the minds of those who lived with her that she was insane. She talked incessantly upon private matters not usually discussed in polite society. She was occasionally guilty of frivolous, objectless acts, such as watering the plants on the chintz pattern of a sofa, running at full speed in public, filling her mouth so full of food and eating in such a manner as to disgust her relations at meals. She had been previously confined three times in asylums, but was fond of alluding to these events in her life, and looked upon the fact of her having been insane as a good joke.

She has left the Asylum several times on leave of absence, and has also been discharged relieved, but has returned at her own request when she felt she was becoming more flighty. She now refuses to leave, although her friends implore her to do so.

Her case may be regarded as one that has just stepped

over the boundary line, but the mental symptoms were very very faintly marked.

II. HYSTERICAL CASES.

Who is there who can approach the subject of hysteria without fear and trembling? Into how many classes and orders is it subdivided? Where do its symptoms merge into actual health, and where into insanity? Of all the cases which have come under the care or observation of the writer, two are remarkably prominent as exhibiting the Alpha and the Omega of the disease.

One case was a country girl aged 16. She was in perfect health, and was possessed of a physique rarely seen amongst the most developed of women. She complained that her legs were swelled, and begged that they might be examined. The circumference of each of the calves was 14 inches. She imagined that she was dropsical, and was somewhat disappointed when she was informed that this exuberance consisted of nothing but muscle and fat.

The other case was more tragic.

A girl with long fair hair used to let it flow down to her knees whenever the doctors entered the wards, and assume a sentimental attitude. Unsatisfied sexual desire was the cause of her illness. She died rather suddenly from pure exhaustion. Cysts of the ovaries and other complications threw a partial light upon the pathology of the case.

The second group includes cases in the hysterical symptoms.

Case 3.—Hysteria with suspicion of relatives, almost amounting to insanity.

Miss D., æt. 30, the daughter of a clergyman; a tall, anæmic patient, with a profusion of black hair, and regular features.

The father had married a second wife, to whom the daughter had taken a great dislike; but the step-mother, as far as could be ascertained, had invariably treated the patient with great kindness.

The catamenia were regular but scanty, the pulse feeble and intermittent, the chest healthy, and the appetite enormous—the patient insisting upon having food of some kind or other ten times during the twenty-four hours.

This young lady had been twice engaged to be married, and on both occasions the marriage had been broken off. She was very coherent in conversation, but excitable, and, although she talked a great deal, said very little to the point. She wished to start for Australia at once, although

unable to accomplish a short railway journey without an attack of hysterical weeping. She proposed she should go to Brighton for change of air, and in the next sentence said she hated Brighton, soon after changing her mind again, and saying she adored Brighton. There were no delusions. The most prominent mental symptom was shown in the intense hatred and suspicion she entertained towards her stepmother. She had a very marked hesitation in speech, accompanied by a kind of gasping sob, which I knew was not present before the attack. The case was treated with bromide of potassium, valerian, ether, iron and camphor water, in the intervals between the monthly periods, and with warm hip-baths, ergot, and aloes when the discharge occurred. Removal from the stepmother was also insisted on. She improved in a few months, and went to Australia.

No medical man would have been justified in signing a certificate in such a case, although the desirability of placing the patient in an asylum was hinted at more than once by the relations.

Hysteria, ending in hysterical mania, the patient being treated at home.

D. T., æt. 21, daughter of a solicitor. Father died of general paralysis, mother very hysterical. Cause of attack, disappointment in love, an engagement having been lately broken off.

For some days before the attack she had almost entirely refused to take food, but she was nevertheless able to take long walks, and skate for some hours daily at a rink. In the evening, attacks of hysteria came on. The family physician was called in, and eventually the writer was consulted, as the case ended in a pure attack of hysterical mania.

The patient, being extremely feeble, was kept in bed. She appeared at first in a semicomatose condition, the eyes being closed. This state lasted a few minutes, and was succeeded by an attack of screaming, mixed with incoherent raving about the beloved one, and complaints of an intense pain in the head.

There were distinct delusions, the most marked one being that the patient was tied down in bed by a complicated network of cords, for which supposition there were no grounds.

Convulsions, evidently of an hysterical character, occurred at frequent intervals.

The patient was fed artificially, and a sedative mixture, containing chloral, morphia, and bromide of potassium, was ordered every four hours. The services of a skilled attendant were necessary.

Recovery was gradual but slow, and there were two or three relapses to the maniacal condition.

When convalescent, the patient was feeble and pale, and spoke in a slow, deliberate, soft tone of voice. She is now perfectly well.

III. CONGENITAL CASES.

Such cases are but seldom brought under the notice of the consultant except in their first stages.

At the magnificent establishments of Earlswood and Normansfield, every type and degree of congenital imbecility is to be found. In private practice it is sometimes a matter of no small difficulty to diagnose a case of congenital imbecility in an adult from one of dementia. The history of the whole life of the patient alone can guide us.

In the upper classes the prognosis is sometimes of high importance in such cases, as frequently the relatives who are spending the money of the supposed imbecile are much alarmed at any prospect of his recovery, which may nevertheless take place in exceptional cases even after years of dementia have been passed in an asylum.

Third Group.

Case 5.—Congenital imbecility. Mental symptoms not sufficiently marked to justify detention in an asylum.

T. C., æt. 19; domestic servant. First-cousin of maternal grandmother had been insane. No other hereditary taint. Six brothers and sisters, all alive, healthy, and sane, except one brother, who died of delirium tremens.

Patient suffered from convulsions at dentition, and has always been somewhat hysterical. Was very backward for her age at school. The chief mental symptom when under observation was loss of memory. If sent to make purchases at a shop she would forget what was required, and return home for information. If she bought anything she would forget to take the change, and leave it on the counter. If sent to sweep the house, she would forget to take the broom. Consequently she has been discharged from several good situations, and considered useless as a servant.

She was employed in housework at home, but, having become more demented, was sent to me by the clergyman of the parish, in the hope that medical treatment might be of service. No drugs were necessary, nor were there sufficient grounds for sending her to an asylum. I could, therefore, only recommend the friends to take her home again, and employ her as much as possible in any work she was capable of performing.

The writer is indebted to Dr. Langdon Down, of Normansfield, for the following most interesting case.

Case 6.—Congenital imbecility. Mental symptoms so prominent at puberty as to necessitate detention in an asylum.

M. P., the daughter of healthy parents, but her mother is highly emotional. Her brothers are all healthy, her sisters more or less delicate, one of them having suffered from strumous disease of the knee-joint. Her mother, when pregnant with M. P., was much distressed by the ill-health of a favourite sister. M. P. was born at the full period, and without any instrumental interference; nor were any artificial measures resorted to for resuscitation at birth. At the age of 2 years it was found that she did not speak, and did not walk. She cut her teeth late, and they decayed very early. She began to talk at 3 years of age, but indistinctly, when it was noticed that her palate was highly arched and her face unsymmetrical. She walked at 3½ years, but for a long time was unable to get up or down stairs alone. She was educated at home with her brothers and sisters, and was then considered to be a reserved but amiable child.

At the age of 15 the catamenia appeared, and a complete change came over her. She became morose, disobedient, cruel, and destructive, pinching and biting those about her, and tearing up her clothes if her wishes were not at once gratified. Gradually fits of violence came on, and Dr. Langdon Down was consulted. He advised her residence at the training institution at Normansfield. Here she soon became more amenable to control. Her reading and writing was improved by tuition, and her taste for music was cultivated. There was, however, a slight relapse at every monthly period, when she became untruthful and pert. After two years' residence she returned home, but, again becoming unmanageable, she was sent back to Normansfield. Her physical health is good; her mental condition unstable, the catamenial period being always marked by insubordination, violent language, rude gestures, and untruthfulness.

The case is interesting as exhibiting the effect of puberty upon a congenital imbecile. Before that epoch, she was sufficiently quiet to be kept at home. Afterwards, she became unmanageable there and a fit inmate of an asylum.

PREMONITORY SYMPTOMS.

These symptoms are of the utmost importance in the preventive treatment of insanity.

They may be divided into three groups.

The first set of symptoms are seen only by the relations, who generally endeavour to persuade themselves that nothing is the matter with the patient.

The second set are seen usually by the family doctor.

The third set come under the observation of the alienist, when suicidal, homicidal, or destructive symptoms induce the relations to throw the responsibility and risk of confining the patient in an asylum upon the consultant.

They may exist for months—even for a year—before any active steps are taken to treat the case medically. Consequently, what might have been a functional disorder, frequently becomes organic, and the friends are then surprised when they are told that the case is most probably incurable.

Fourth Group.

Case 7.—Premonitory symptoms of insanity. Premonitory symptoms, not ending in mental derangement.

Mrs. R., æt. 35; a professional singer; wife of an artist.

Three years ago, the patient was under my care. She was at that time suffering from an attack of puerperal mania, followed by dementia. Since then she has had a fourth child, without any mental disturbance.

She is stout and healthy-looking. Quite regular, but there is not sufficient catamenial discharge. At these periods she suffers from a severe 'rumbling' headache in the cerebellar region, great irritability, as shown by her unnecessary abuse of servants and children, and loss of memory, evidence of which is displayed by a total forgetfulness of appointments, of the dinner-hour, and by omitting to make certain necessary purchases for the house. These symptoms only occur at the monthly periods. She is herself aware of them, and dreads another attack of insanity.

Chest healthy. Fine expansion, but heart's action very feeble. Ordered iron and ether, and to come back for advice just before the menstrual period.

January 21.—Four days before this critical time. Ordered warm hip-baths and ergot. Pil. Aloes et Ferri every night.

This treatment was carried out on two occasions, at a month's interval, with marked benefit. The patient became more cheerful; there was no further loss of memory, and she was able to appear and sing at several public concerts without inconvenience.

This case forcibly illustrates the importance of early treatment. The patient had suffered from a first attack from the neglect of the husband to take medical advice. On the second occasion, being warned by previous experience, he brought his wife to be treated the moment the old premonitory symptoms appeared. The attack was doubtless staved off by early treatment. This case is to be regarded as one outside the pale of insanity, as the mental symptoms were not sufficiently marked to allow the patient to be placed under medical certificates.

Case 8.—Premonitory symptoms of insanity sufficiently marked to justify detention in an asylum.

Mrs. P. E., æt. 46. Sent to consult me by Dr. Ackland, from Bideford, in Devonshire. The patient was seen immediately after a long journey, and appeared much fatigued. Had undergone several operations for fibrous tumour of the uterus. Catamenia, regular and profuse, every two or four weeks.

She was very irritable, and slept badly. Her memory was much affected. She had a fear that she should drown herself, and whenever she saw a knife, begged her relations to take it away, in case she should commit suicide.

The patient was surrounded by watchful sons, daughters, and servants. Her character was weak, and I had no fear whatever that she would carry out her suicidal intentions, if ordinary precautions were taken to prevent it.

I therefore advised that she should return home, and that a skilled attendant should watch her at night, the daughters undertaking never to leave her alone by day.

Having taken such a long journey, she thought it necessary, however, to consult more than one doctor. She therefore went to a well-known alienist in London, who immediately called in two practitioners. Certificates were signed, and she found herself sleeping that night in his private asylum. She remained there a week, but, being a very sensible patient, became so much distressed at being in an asylum, and at being separated from her friends, that she was removed to her home, where the plan of treatment I had originally recommended was successfully carried out.

In this case the patient had decidedly overstepped the boundary line. There was no doubt that she was insane. Yet the symptoms were so slightly marked that the case affords a good example of one where sanity has only merged into insanity.

CONVALESCENT CASES.

Cases of convalescence from insanity are seen but seldom by asylum superintendents, except, indeed, at St. Luke's Hospital, where the patients frequently present themselves to the superintendent, with whom they are familiar, for examination, in preference to being overhauled by a strange doctor.

In almost all cases, three months after discharge, there are still traces of the old disorder.

The patient looks somewhat lost and distressed, the angles of the mouth are drawn down, there is a general feebleness about the outlines of the features, and the bodily health is never quite re-established. They complain, moreover, of

irritability, although they are aware it is due to their disease. They are sometimes easily moved to tears, and their letters express eloquently their undecided and vacillating state of mind. 'I am sometimes sad, and sometimes happy,' writes a patient. Another always places the sign of the cross and the letters I.H.S. outside and inside her otherwise sensible letters. These traces of the old disorder are trivial in the extreme, but are not without interest, especially if the physician has taken a personal interest in his patient.

Fifth Group.

Case 9.—Convalescence from insanity. Mental symptoms not sufficiently marked to justify detention in an asylum.

Mrs. A. B., æt. 50. She had been insane for fifteen months, and had been transferred from one private asylum to another on four or five different occasions. This was a most difficult case. She was received when her mental symptoms, if any, were so slightly marked, that no one, seeing her for the first time, could possibly have pronounced her insane. That she had been insane there was plenty of evidence to prove, but she came under observation only when convalescent. Being also a very clever woman, she knew well how to conceal any symptoms which might point to insanity. I had several long conversations with her. She was well read. She was an implicit believer in spiritualism, but, as she argued, this alone did not constitute insanity.

She disliked being confined in an asylum, yet had a vague dread that if she left she would be hidden out of sight somewhere by her husband. She was always in a very unsettled state of mind, one day saying she wished to go on leave of absence, and on another that she would rather remain where she was. She was very untidy in her habits, and was also in a constantly morbid, restless condition, passing rapidly about the house from room to room, without any apparent object. After a month's residence in the asylum, she was discharged recovered.

Case 10.—Convalescence from insanity. Mental symptoms very faintly indicated.

Continued residence in an asylum, by the express wish of the patient herself.

Mrs. C. F., æt. 50; admitted to Otto House on June 28, 1862.

This lady, to all appearance, is perfectly rational in conduct and conversation. She acts as a companion to the other more insane patients in the house. She is constantly employed in gardening and looking after her numerous birds and other pets. The mental symptoms are indeed but

slightly marked. She grumbles occasionally because she is not allowed an excessive amount of stimulant, and is somewhat hypochondriacal, always fancying there is something the matter with her.

If watched when she is alone, she is seen to throw up her eyes and to gesticulate, and these movements are possibly connected with some insane idea. She has but one delusion, which has remained fixed for many years, that if she were to set her foot outside the door of the establishment, her children would be eaten up by tigers. Consequently she never leaves the house, and refuses to accompany the other patients to the seaside in the autumn. Her husband is a clergyman. Since her admission he has gone to a new living, where nothing would have been known of this lady's having been incarcerated in an asylum. The husband, and everyone connected with the asylum, at that time made every effort to induce her to leave, thinking that then, if ever, she might be persuaded to go to a new home. These efforts were entirely unavailing, and she now resides in the establishment at her own free will, although the mental symptoms are too marked to allow of her becoming what is legally known as a boarder.

In this case, convalescence, up to a certain point only, had been established. It may be considered as one in which the patient still remains within the boundary line of insanity.

CLINICAL NOTES

ON

CONDITIONS INCIDENTAL TO INSANITY.

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I.—ON THE TREATMENT OF DIRTY HABITS IN THE INSANE.

THERE is, perhaps, no more troublesome and annoying condition incidental to the progress of diseases of the insane than palsy of the emunctory sphincters. Dirty habits constitute one of the most repulsive features of patients whose whole mental and bodily state would cause disgust were that feeling not neutralised by pity. Such patients are involuntary agents in thwarting the best endeavours of attendants and others in their efforts to promote cleanliness in their wards, and a sense of homeliness in the patients associated with them. Not only so, but even though the patients themselves may have sunk below the entertainment of any sense of discomfort, yet the persistence of incontinence in the operation of the emunctory functions occasionally shortens their lives by the production of such bedsores as are sufficient to add irritative fever to the sum of the miseries with which they are burdened. Attention was

specially directed to this condition during a period when by a remarkable accumulation in this asylum of patients suffering from extreme dementia, associated with general paralysis, destruction of brain substance by clot or degeneration, and of others in the deepest stage of senile dementia, prominence was given to the difficulty of maintaining a proper degree of cleanliness in the wards assigned to such patients.

A hint as to what might be done in the way of counter-acting paralysis of the rectal sphincter and its associated muscular fibres, was obtained by the action of strong suppositories of tannin on a very demented patient, who both before and after the opening of an ischio-rectal abscess was excessively dirty. After the use of several 10 gr. suppositories—one having been used nightly—he became quite clean both by night and day. On subsequently examining the condition of the rectum and anus in several sleeping advanced general paralytics, it was found that by great relaxation of the sphincter during sleep, the contents of the rectum were simply permitted to pass out in a continuous manner, and it seemed reasonable that if by any means even a limited amount of tonicity could be restored to the sphincter, a certain check would be placed on this form of incontinence. The principle appears somewhat mechanical, but as it is a physical principle operating through nature for the production of beneficial results not otherwise attainable, it cannot altogether be despised. Accordingly, eight of the dirtiest patients on the male side of the asylum were selected for a trial of the treatment. Most of these patients rarely, if ever, passed a night without voiding their motions in bed, and not a few of them required to be changed on that account more than once during the nine hours the night attendants were on duty. Almost immediately the result was found to be satisfactory. In the instance of two of the patients—one of whom was looked upon as the dirtiest case of all—the dirty habits ceased from the very first day of the use of the suppositories. These were employed every night for a month, and during the subsequent twenty-eight days special notice was taken of these two cases, with the result of determining that this successful issue was maintained during the whole

of that time. As evidence that the patients did not in any respect suffer from the treatment, it may be said that both are alive and in much better physical condition than when they first had the suppositories. Though in the other six cases the benefit was not so strongly marked, yet it was sufficiently great to merit attention. Thus on the sixth night of the use of the drug not one of the selected patients was dirty, and this satisfactory state of matters was repeated on other four out of the thirty nights over which the trial was extended. At the end of the thirty days it was ascertained that the aggregate sum of the occasions on which these eight selected patients had been dirty was forty-eight, giving an average of 1.6 times a night for eight of the most degraded male patients in the asylum. Or, putting it somewhat differently, only a fraction more than two out of sixteen otherwise incorrigibly dirty patients continued so after the employment of the tannin suppositories. To determine, also, the permanence of the action (though little good was anticipated in that direction), notes were carefully taken for twenty-eight nights following the discontinuance of the suppositories. On two nights out of the twenty-eight all the patients were clean, and the aggregate sum for these twenty-eight nights reached only fifty-six, or, in other words, the average number of the eight men, dirty each night of the twenty-eight, was 1.86. There is another method of estimating the good result obtained by the use of the means under discussion. In a ward containing fifty-two patients, the supplementary wash, that is the wash sent to the laundry as being dirty before the stated time for changing, consisted at the commencement of treatment of sixty-one sheets, eighty-six shirts, and fifty-two blankets for one night. At the termination of the treatment it had been brought down to thirty-seven sheets, forty-five shirts, and thirty-one blankets, representing a daily saving of wear and tear, and labour of washing of twenty-four sheets, forty-five shirts, and forty-five blankets from one ward alone.

During the use of the suppositories, several collateral observations were made. It was found that not only during the night, but also during the day, the condition of the

patients was improved. They were less frequently dirty and still not constipated, and it was frequently seen by the charge attendant and others that members of the group who formerly used to pass their motions under them, went voluntarily to the closet. This appeared to a certain extent to justify the conclusion that an increase of local tone of the sphincter and other muscular fibres of the rectum had sufficiently increased the capability of appreciating local irritation as to induce an automatic, if no higher, action on the deranged intelligence, and had led to the mechanical performance of an action in a manner imprinted on the mind as one of the earliest records of infantile education.

It would be impossible to quit this subject without referring to the influence which this treatment has had in the prevention of bed-sores. At present the state of matters is that whereas, amongst the class of patients from which the eight referred to were selected, bed-sores used to be not uncommon, such a thing as a bed-sore is almost entirely unknown, and the only cases which for months have occurred on the male side of the Asylum existed when the patients were admitted. Several agencies have been operative in the production of this result, but certainly the capability of maintaining cleanliness in the most demented patients is not the smallest. In concluding these remarks it is only necessary to quote from the record of a case which may subsequently be published for other purposes :—

J. B. *July 24, 1876.*—He has become very dirty and degraded in his habits. Two days ago he was seen to have a hard swelling of both sides of the scrotum, associated with fever and local heat and vascularity. The condition was one of orchitis. The swelling rapidly increased, and assumed alarming dimensions. Before applying leeches, it was seen that a black-coloured bleb had appeared on the lower part of the scrotum. This morning gangrenous action was observed to be spreading from this spot. For some time before going to bed the patient (who is an advanced general paralytic) had been dirty both night and day, passing his motions wherever he might be; and this adverse condition was still existent at the commencement of the gangrene. The heavy scrotum was held up by a towel stitched to a bandage round the abdomen; still, as he was constantly interfering with the bandage and the charcoal poultices, the contents of his rectum were constantly coming in contact with the gangrenous surface. Tannin suppositories were ordered night and morning, and an occasional injection or dose of castor-oil. In a few days about the whole of the lower third of the scrotum

sloughed off, and the lower half of both testicles was exposed. The poultices were soon changed for carbolic lotion and oil.

In about a fortnight healing action was pretty well advanced, and the edges of the curtailed scrotum were drawn together by sutures. In another month the line of union was complete. After the employment of the tannin suppositories he ceased to be either wet or dirty, and though they were employed only for about a week, he has been cleanly in his habits ever since.

It is difficult to see how in such a case so good a result could have been obtained, if the incontinence of the bladder and rectum had not been overcome. It would have been impossible also to use constitutional remedies without prejudicing either his physical or mental ailment, so that there can be little doubt that in this case the use of tannin suppositories was of great temporary benefit as regards the gangrene, and considerable subsequent benefit as regards the patient's comfort during his progress through the later stages of an incurable disease. There is some reason to believe that these suppositories not only increase the tone of the *sphincter ani*, but by associated action have a beneficial effect on the *sphincter vesicæ*, inasmuch as when incontinence of urine and want of control over the bowel have been present in the same patient they have more than once been simultaneously cured.

The suppositories were made according to the following formula :

R Acid. Tannici, grs. 120.
 Adipis Benzoat., grs. 80.
 Cere Albæ, grs. 20.
 Ol. Theobromæ, grs. 140.

Dissolve the oil of Theobromæ with the wax over a water-bath, then add the tannic acid and lard, previously mixed in a mortar. Pour the mixture while fluid into suitable moulds, capable of holding 30 grs. each.

Mr. G. W. Bracey, the apothecary to the West Riding Asylum, informs me that if in making the suppositories the mixed white wax and oil of theobromine be too warm they do not combine well with the other ingredients—the latter falling to the bottom. If, however, they are combined at the proper temperature, the mixture can be heated without deterioration for purposes of subsequent manipulation.

II.—VORACIOUS APPETITE AS AN INITIATORY SYMPTOM OF BRAIN TUMOURS.

ON examining the record of symptoms of those cases of Tumour of the Brain which have supplied pathological specimens for the Museum of this Asylum one condition is found which at first was regarded as a mere coincidence, but which by repetition came to claim some consideration. In many of such cases it is recorded that at the commencement, or during the progress, of the disease, the appetite was 'ravenous,' 'voracious,' or 'very good.' It is important to observe that such notes, in the instances which are to be adduced, were not made when the patient was demented, inasmuch as voracity is so commonly associated with extreme dementia that the phenomenon would lose its value if it occurred only in the latter stages of cerebral disease. One thing also must be taken into consideration, namely, that the observations made regarding these cases were purely retrospective, and were made during a summary of cases for pathological purposes, in most instances several years after the death of the patients, and by one who had had no communication with them during life, so that no preconceived idea could possibly modify the correctness of the observations. No attempt will be made to explain the condition indicated, but only such evidence as exists in our records will be advanced, so that others may have an opportunity of determining whether or not voracity constitutes a common symptom of the early stages of cerebral tumour growth.

M. S. Tumour, part of which attached to the vaulted aspect of the parietal lobe, had a fibrous covering of a dirty white colour, and was of soft consistence, the remaining part being separated from that behind it by a constriction, and consisting of a pulpy red substance held together by a fibrous matrix, and lying in an excavation in the body and part of the right wing of the sphenoid bone. The ethmoid was broken up, and the osseous substance softened, and in some places caseous. Part of the tumour projected into the Sylvian fissure, and was hard, and in places calcareous.

The patient had ptosis of the right eyelid. The tongue was protruded to the left, and the mouth was drawn to the same side. Her vision was defective. She said that *some time ago her appetite was voracious*. There was a certain amount of loss of power of the left side of the body, and a

corresponding weakness of sensation. The right eye-ball was prominent, and the left pupil was contracted and irregular.

M. F. In the right hemisphere, occupying the parietal lobe, there was a tumour the size of a small orange, which projected visibly from the brain mass. It occupied the postero-parietal lobule, the angular gyrus, and the upper extremity of the first tiers of the temporo-sphenoidal gyri. It was of a dull purplish colour, and not sharply defined from the surrounding cerebral substance, but merged into it by imperceptible gradation. The lower half resembled most the cerebral tissue in colour, but was harder than it. The upper part was quite pulpy. There was no wasting of the convolutions, which were firmly compressed. The optic nerves were somewhat wasted.

The patient had first severe and almost fatal epistaxis, then violent pains in the head and temples, accompanied by vomiting. *From the beginning of his illness he manifested a ravenous appetite.* 'He was always wanting to eat, and never seemed satisfied.' Strabismus, inequality of the pupils, loss of memory and sight, and great physical prostration followed, and finally he lapsed into coma, which was preceded by twitchings of the left side, and died.¹

A. P. On cutting into the right cerebellar hemisphere, its proper substance was found to be almost completely converted into a hard fibrous mass, apparently of the nature of scirrhus.

This case was complicated by the existence of extensive cerebral wasting. The symptoms were those of general paralysis, but the patient also dragged her right leg on walking. She had a certain amount of dementia, which soon became much more marked, but from the first it was noticed *that her appetite was ravenous.* She developed complete right hemiplegia, and protruded her tongue to the right.

S. B. *Carcinoma cerebri.* The whole of the convolutions of the left parietal lobe, as well as the ascending frontal and the posterior extremities of the three frontal gyri had an expanded and flattened appearance, as if they had been altered in contour by some force acting from within. These convolutions had a dense consistency, and together formed a tumour, bulging to the extent of fully an inch beyond what would have been the proper outline of the hemisphere.

On admission, it was said that the patient's illness began with a succession of strokes, in which he did not lose consciousness, and which deprived him of the use of his right arm and right eye. While in bed he had more 'strokes,' and became much excited. He showed no loss of memory, but was somewhat embarrassed in his expression of ideas. What he did say, however, was rational. For two months after his admission there is written evidence that he had little or no dementia. His speech and power of deglutition gradually failed him, and he died in the fourth month after admission. On admission it was specially noted that *'his appetite was good.'*

Though this phrase is not so striking as the terms used in the cases just quoted, yet it commands a certain amount

¹ This case is fully reported by Professor Ferrier in his paper on 'Pathological Illustrations of Brain Function.'—'West Riding Asylum Medical Reports,' vol. iv. p. 36.

of attention, inasmuch as it contrasts strongly with what would have been the state of the appetite if a cancerous tumour of the same size had existed in any other part of the body.

In another case of tumour of the right orbital lobule the patient is also said to have taken his food well, but the case is not sufficiently fully detailed to justify its use under this head. The fact that the state of the appetite in other cases of brain tumour has not been recorded, is no evidence that voracity did not exist. The condition might be readily overlooked in an asylum where, amongst many demented, ravenous appetites are the rule; and the fact that without any preconceived notions attention was, under the circumstances, directed to the existence of the condition in so many instances of a comparatively rare form of disease, favours the probability that voracity has passed unobserved as an early symptom of brain tumours.

III. RECOVERY FROM INSANITY IN CASES WHERE HÆMATOMA AURIS HAD OCCURRED.

As the existence of Ot-hæmatoma, or the insane ear, is commonly looked upon as indicative of incurability in insanity, the following cases have some value.

C.B. Admitted June 16, 1875. In March of the same year the patient had rheumatic fever with pericarditis, and was dangerously ill. For a considerable time he was very weak and unable to follow his employment, and consequently was somewhat depressed. About 10 days before admission he suddenly became excited. He refused to go to bed, was restless at night, wandered about the house, and threatened injury to his wife and those who were restraining him. He was noisy in his conduct and irrational and incoherent in his conversation. He made a suicidal attempt to get through a window, and destroyed a clock and other articles of furniture. He had delusions that he was filled with vitriol, which had been thrown at him by members of his family, and in consequence threatened them with punishment. He was dirty in his habits. He had been sober and industrious.

On admission he was restless, timid, and, when much irritated, combative. He had almost to be forced to take his food. He could not sleep without narcotics. His expression was stupid and absorbed. He understood questions, but answered them irrationally and incoherently, and though he was evidently full of delusions, their nature could not be ascer-

tained, as he lapsed into dogged silence during the progress of his examination. He was in bad bodily condition, and had chronic pericarditis. When his case was taken, mania was diagnosed and the prognosis was recorded as grave. He was treated for the heart affection.

The active excitement gradually diminished, but stupor increased. He took only liquid food, and that by hand-feeding. A fortnight after admission he is said to have been much worse. He was exceedingly timid, and showed intense nervousness when anyone went near him. Digitalis, which had been given for a short time and stopped, was resumed, but without effect. Up to October 1875 he continued in the same state of insane timidity and restlessness. At this time he was a picture of misery. When he was approached by anyone he edged away in a half-guilty, half-frightened manner, and if a hand was placed upon him he scratched it virulently, and the best-intentioned movements caused him to lift up his elbow in a cowed and terrified manner, as if to protect his head. He had frequently to be fed with the stomach-tube. He could not speak a rational word, and avoided as much as he could the presence of the other patients and attendants. Chloral was ordered, and produced no lasting effect. *He now had insane ear of the left side.* Chloral was discontinued, and $\frac{1}{4}$ grain of hyoscyamine ordered 3 times daily. At first, as was to be expected, the stupor again gave place to excitement, which continued till the drug was stopped 5 days afterwards. After that his excitement ceased, but he continued stupid till February of the present year, when he began to get much better. He continued to improve, took voluntarily to ward work, and subsequently requested to be employed at his own trade as a mechanic. He worked most industriously at the most intricate parts of his trade, became quite sharp both in movement and in conversation, and, instead of avoiding everybody, made himself prominent in the promotion of amusement and good feeling. When discharged, in May 1876, he had marked traces of the insane ear, the external organ being shrivelled up into a deformed knot, and when asked how he would explain this condition to inquirers, he said cheerfully that he would do it in the best way that he could.

For the notes of the following corresponding case, I am indebted to Dr. Cameron, Superintendent of the Argyll and Bute Asylum.

A. C.; admitted February 25, 1876; discharged recovered, July 24, 1876; occupation, slate-quarrier; age, 27 years; single. The patient had been insane one month before admission. The exciting cause was unknown, but the patient's mother was insane, and died in the County Asylum.

The patient's brothers informed me that he had been 'a very good young man,' and had taken a leading part at prayer-meetings and Sabbath-schools. He had refused food now and then before admission, but never for a longer period than 24 hours at one time. On admission, he was emaciated, but otherwise in tolerably good bodily health. Mentally, he was excited, and talked almost incessantly, quoted passages of Scripture, prayed, 'exhorted,' declared himself to be Lord Nelson and a minister of the Gospel. He re-

remained in this condition about six weeks, when he began gradually to improve. Two months after admission, and after decided improvement had manifested itself, hæmatoma of the left ear was observed. The tumour was about the size of a filbert, and never became larger. It gradually shrank away, leaving the ear deformed, but not to a great extent. Patient at this time was very taciturn; never spoke, except when addressed. He was sent out to work in the farm about a month after admission, and continued to work out of doors subsequently until he was discharged. During the last month of his residence in the Asylum, the patient became more communicative, though he continued to be rather reserved in manner.'

Dr. Cameron kindly communicated with the patient's father and medical man a month after his discharge, and found that A. C. had been at work every day since his discharge, and that, with respect to both mental and bodily condition, he was as well as ever he had been.

IV. ON PHOSPHORUS IN THE TREATMENT OF DEMENTIA.

The importance ascribed, first by somewhat vague surmise, and latterly by scientific analysis, to phosphorus as an important element in the structure of the brain-tissue suggested the idea of trying to what extent that substance might be useful in cases of dementia where there appeared to be evidence of defective brain-nutrition. Observations on the matter have extended over five months, but unfortunately the results are purely negative. The drug was given in the form of coated pills, each containing gr. $\frac{1}{16}$ of pure phosphorus. One pill was administered night and morning. In the first case the patient showed symptoms following upon a condition which at first resembled acute dementia but which in course of time varied from the typical features of that disorder. When working in the grounds he eluded the vigilance of the attendant, and was found suspended by the neck-tie from a nail driven into the wall. Subsequently, he was very stupid, would not or could not answer a single question, and sat constantly in one place unless removed from it. At times also he tried to strangle himself with his neckerchief. On May 18, 1876, he was ordered to have gr. $\frac{1}{16}$ of phosphorus in pill night and morning. On May 29, it was noted that there was great improvement. He was much brighter in his expression, answered simple questions, and showed more animation. On May 30 he was much quicker in his movements, and answered questions readily.

His circulation was exceedingly languid. He now began to make himself useful as a ward-helper, and was comparatively cheerful and communicative. He was certainly less demented, and had improved in bodily health, but he still manifested great weakness of mind both in his talk and his actions. On June 2, it was noted that he had made no progress since last report, and on the 26th it was recorded that all the patients under phosphorus were mentally unchanged, except the one under consideration, but that all of them were gaining flesh. On July 8, it was again noted that the patients were unimproved with this one exception, but even he made no advance beyond the point which he had reached during the first fortnight of treatment; and though the drug was given to him for several months longer, no further advance was made by him, and he still remains fatuous and somewhat slow in his movements, but has continued to make himself useful in the wards, and has entirely abandoned his suicidal propensities.

In this case alone was even a modified satisfactory result obtained by the use of phosphorus. Of the other cases tried, two were patients labouring under dementia consecutive on mania, and one was a case of dementia following on acute melancholia. In the two former the prolonged use of the drug caused no mental change whatever, and in the latter case, though the patient once gave hopes, by whistling cheerfully for a whole day, that the phosphorus was about to produce a modification of his symptoms, yet next day he was as dull and stupid as ever, and now gets gradually more and more demented. In still another case of consecutive dementia the improvement during the first week of treatment was very marked, but this change was soon followed by a relapse. Another period of progress followed, but was equally transient, and now the patient is in much the same state as when the phosphorus was first given, and the use of the drug has been abandoned. It is noticeable that the only patient who underwent improvement did not alter in weight; whereas those who did not improve, gained from 12 to 35 pounds in the course of 4 months. It is the negative part of the observation which is of value, as it is a common thing

for demented to increase in weight without the intervention of phosphorus or any other drug.

V. EPILEPTIFORM SEIZURES.

Epileptiform seizures in the course of general paralysis of the insane are so common and so widely appreciated as part of the phenomena of that disease, that it would have been unnecessary to refer to them, unless for the purpose of mentioning a case in which epileptiform fits were exceedingly numerous. Advantage is also taken of the case to refer to the almost invariably beneficial action of hydrate of chloral in such paroxysms as those about to be described. It is amusing to see, in the weekly and other journals, occasional announcements of the fact—as if it were an original discovery—that chloral hydrate is excellent in warding off convulsions. For the past three years at least, chloral hydrate has been by many recognised and used as the only drug necessary to cut short the epileptic status, and to put an end to the convulsive twitchings and contortions of general paralysis. The question was so fully entered into by Dr. Wallis in a paper ‘On the Therapeutic Value of Chloral Hydrate in Epileptic Convulsions,’ published in the last volume of these Reports, that it is not necessary for me to say more than that chloral hydrate is perhaps the most useful medicine which has ever been employed in asylum practice. The benefits resulting from its use are so clearly and quickly perceptible, and the dangers resulting from the employment of a good preparation of it are so few, that it constitutes a most valuable item in the alienist’s pharmacopœia.

G. H. was a general paralytic, whose disease seems to have begun about October 1873. In July 1874 he had his first convulsive attack, which affected chiefly his right arm and the right side of his face. In October he had another attack, in which the right arm and leg were convulsed, and which was stopped by 40 grains of chloral. After that bout he had no more till May 1875. On the 17th of that month he began to have unilateral convulsions. Forty grains of chloral had no perceptible effect. Next day, the fits continuing, he had 40 grains of chloral; but this dose only diminished the severity, and apparently not the number, of seizures, but in 2 hours he fell asleep, and slept for an hour. About 8 hours after the administration of the 40-grain dose, he had 70 grains—26 fits having occurred in

the interval. He had 1 more fit, and fell asleep, sleeping for 5 hours. Next morning at 9 he had another drachm dose, had 2 fits, and then slept for $4\frac{1}{2}$ hours. He had another drachm dose at 9 P.M., and again at 9 A.M., of the following day; but from this time forward the number of seizures increased with alarming rapidity, and chloral seemed to lose all control over them. He dozed for short times at long intervals, but for the most part went out of one fit into another. It became necessary to push the use of the chloral to the utmost. Repeated 1-drachm doses were given, but only with the effect of reducing the severity of the fits. Between 10 and 11 P.M. of the fourth day of the seizure he had 33 fits, and one and a half drachms of chloral were given, and he slept for 3 hours. He was still conscious between the fits. On the morning of the ninth day of the seizure, even doses of one and a half drachms of chloral failed to arrest the convulsions. They gradually increased in number, till he fell into a comatose state and died, having been convulsed for 15 days. During that time he was seen in 1,849 separate fits.

It will be seen in this case how necessary it is to give doses of chloral sufficiently large to counteract the central irritation. Here forty grains was an insufficient dose, and even one drachm was only of benefit towards the commencement of the seizure. Then when that dose had repeatedly failed, a drachm and a half was as potent as one drachm had been at an earlier stage. In fact it is in such cases impossible to say what a dangerous dose of chloral is, inasmuch as the best proof that a dose has been insufficient is that convulsions occur after its operation, and it appears to be a sound principle that it is safe to continue giving chloral as long as convulsions exist for the drug to counteract, unless it is seen to cause impairment of the movements of the chest.

In the case recorded it was found that, in the meshes of the pia mater, and on the second frontal convolution about an inch from its posterior termination, there was a small irregular bony nodule about the size of a pea. The arachnoid was thick and cloudy. The frontal convolutions were much wasted, and the parietal less so.

In the discovery of such a constant exciting condition, it ceased to appear wonderful that chloral had been useful in allaying the convulsions only temporarily. Experience daily shows that in most uncomplicated cases of epileptiform seizure, the drug acts with a certainty which brings conviction to the most incredulous observer.

VI. ON THE STATE OF THE PUPIL AS AN INDICATION OF CERTAIN PHYSICAL PHENOMENA IN THE INSANE.

The conditions of the pupil as to size, and reflex excitability of the sphincter and dilator of the iris to the stimulus of light, have long been regarded as elements in the symptomatology of cerebral disease; yet, undoubtedly much obscurity still involves this subject, and more especially with regard to the relative value and significance of these signs. The subject is one of peculiar difficulty, owing to the complex arrangement of the nervous apparatus of the organ of vision. I trust a few observations which I have made recently relative to certain oculo-motor derangements in insanity and other affections, may not be entirely devoid of interest to the readers of these clinical items.

The reflex arrangements by which the varying dimensions of the pupil are regulated, depend, according to general acceptation, upon the second, third, and fifth cranial nerves, and a branch from the great sympathetic system; the ciliary ganglion receiving from this supply its sensory, sympathetic, and motor roots. Of these the optic nerve and ophthalmic branch of the fifth nerve stand in the relationship of centripetal nerves while the third and sympathetic, constitute the motor supply for the iris. Can we, however, rest satisfied with this scheme as representing all that is requisite to explain the varying dimensions of the pupil in health and disease? Certainly so far as the mere special or essential adaptations for the requirements of vision are concerned it is sufficient; yet it will be apparent, on more careful examination, that the question when more generally considered becomes one of much greater complexity. The reflex relationships existent between the great sympathetic system and the purely sensori-motor strands of the cerebro-spinal axis suffice to explain the wide range of influences which may thus be brought to bear upon the action of the dilator muscle of the iris, and it is to this more general reflex scheme which I wish more particularly to call attention.

Effect of Sensory Excitation.—I find that irritation of the peripheral distribution of any spinal sensory nerves from the first cervical to the last sacral, as also of the trigeminus, is invariably accompanied by a dilatation of the pupil. Thus pricking the integument with a needle or other irritations will be immediately followed by a change in the width of the pupil. On reference to authorities upon these points, I find it stated by Claude Bernard that he had noticed dilatation of the pupil on pinching any branches of a sensory nerve from the sciatic to the fifth in animals, and that this action of the iris occurs simultaneously with the expression of pain. It is impossible not to appreciate the importance of this observation with respect to the pupillary modifications due to varying physiological and pathological conditions.

Now, it is well known that irritation of any sensory nerve is invariably followed by vaso-motor disturbances.¹ The arterioles of the vascular area, nearly corresponding to the distribution of the nerve, undergo a marked dilatation, whilst all other portions of the circulatory system exhibit a notable rise in arterial tension. The theory of inhibition has long accounted for these changes in arterial tension, and the arguments for the existence of such inhibitory centres, whether intra-spinal or intra-cranial, have been most forcibly advanced in an Article by Dr. Lauder Brunton in the 'West Riding Asylum Reports.'² Latterly, however, this view has been strenuously combated by Schiff and Goltz,³ who by numerous experiments have sought to explain what was originally regarded as inhibitory, as being really due to the existence of active dilating nerves, the vascular dilatation being regarded as an active, and not a passive or paralytic condition.

Whether the theory of inhibition or that which has been termed the 'Duality of the Vaso-motor System' be established as the explanation or not, the fact remains that irritation of any cutaneous sensory nerve, whilst causing dila-

¹ 'Handbook to the Physiological Laboratory,' 1873, pp. 242-6.

² 'On Inhibition, Peripheral and Central,' 'West Riding Asylum Reports,' 1874.

³ 'On Dilating Vaso-motor Nerves,' 'Pflueger's Archiv,' ix. 475.

tation of arterioles throughout its own area of distribution, is accompanied by quickened cardiac action, elevation of arterial *tonus* elsewhere, and dilated pupils.

Effect of Motor Discharges.—Having so far followed out the connection between a dilated pupil and an irritation reflected along the sensory strands of the cord to the sympathetic, I would advance another proposition, which, from a clinical point of view, I regard as one of importance, and one which I do not believe has been recognised before. It may be thus expressed:—*Certain psychico-motor discharges are invariably accompanied by a dilatation of the pupils, both in conditions of healthy and of abnormal activity of the nervous centres.* For some time past I have recognised this fact, and on pursuing the subject further it became apparent that the voluntary movements of the limbs which are accompanied by dilatation of the pupil are not so much those of simple contractions of single muscles, the effect of which is apparently *nil*, but, rather the more complex movements of several muscular co-ordinates. Those movements of the mouth and limbs in man and animals devoted to prehension, mastication, and progression, affected the pupil similarly, whilst, on the other hand, I have always failed to elicit the same result during the most vigorous activity of the muscles of articulation. Now, this fact appears to me to be one of special interest, and for this reason. Those movements of the eyeball which assist in what may be aptly termed progressive vision, viz. convergence and divergence, are of course invariably associated with contraction and dilatation of the pupils. Dr. Hughlings Jackson, when alluding to the specific distinctions between the upward, downward, lateral, and rotatory movements of the eyeball and those of convergence and divergence, has clearly indicated their relationship, by stating that whilst the former are subservient to *tactile* vision, the latter assist merely in that *progression across space* which vision requires, and the centres of which are probably cerebellar.¹ It is interesting in this light to regard the diversified movements of the eye, mouth, and limbs which

¹ ‘Observations on the Localisation of Movements in the Cerebral Hemispheres, by J. Hughlings Jackson, ‘West Riding Asylum Reports,’ 1873.

are subservient to prehensile and progressive or locomotor movements, as involving in themselves also subordinate and more automatic actions, of which these movements of the pupils may be taken as an illustration.

The doctrine of evolution as applied so lucidly by Herbert Spencer to the developmental history of the nervous system demands from us the dictum that all highly integrated or evolved mental processes should include, or, in other words, represent, the sum-total of all the lower and more automatic. When, therefore, we call to mind the constant associated movements we have been considering, of progression as applied to the limbs and the eye, and the constancy of their mutual or synergic action, and regarding the pupillary modifications as a necessary accompaniment of the ocular adjustments for near and distant vision, it becomes apparent that the dilatation accompanying the movements of the limbs might probably be referred to Darwin's first principle of 'Associated Habits.'

Returning once more to the affective side of mental life, we must not neglect to consider the influence of the more *highly evolved sensory* endowments on the pupil.

Effect of Emotional Excitation.—We have seen how centripetal currents originating in the peripheral cutaneous nerves of sensation affect the pupil reflexly through the oculo-spinal tract and cervical sympathetic; but, subjective sensations and the various still higher emotional states have a like influence. Gratiolet¹ affirms that the pupils are always contracted in rage; and undoubtedly the vascular disturbance such as the blood-shot eye, protrusion of the eyeballs from engorgement of the head with blood, may, by affecting the retinal sensibility, lead reflexly to contraction of the pupil, but I have no hesitation in stating that the *primary* effect of anger or rage on the pupils is quite the reverse. I remember many instances proving this to be the case, and especially so but a few months ago when examining a sullen, morose epileptic indisposed to submit to any interference; after each question his reply was a most

¹ 'De la Physionomie,' 1865, p. 346.

expressive scowl, his eyes flashed with rage, whilst a momentary but *extreme* dilatation of the pupils occurred each time.

Of the depressing emotions there cannot be the slightest doubt but that fear up to its extreme of horror, and sorrow to its extremes of anguish, have as a frequent accompaniment permanently dilated pupils. The Melancholic wards of our asylums teem with such instances. Darwin has devoted a short paragraph to this subject in his work on the 'Expression of the Emotions,' and theorises as follows on the origin of this connection:—'No doubt the fears of man have often been excited in the dark; but hardly so often or so exclusively, as to account for a fixed and associated habit having thus arisen. It seems more probable, assuming that Gratiolet's statement is correct, that the brain is directly affected by the powerful emotion of fear, and reacts on the pupils; but Professor Donders informs me that this is an extremely complicated subject. I may add, as possibly throwing light on the subject, that Dr. Fyffe of Netley Hospital has observed that in two patients the pupils were distinctly dilated during the cold stage of an ague fit. Professor Donders has also seen dilatation of the pupils in incipient faintness.'¹ It will, I think, be more readily understood when we reflect that the emotions are but highly evolved states of common sensation which was the primary basis or substratum out of which they originated; and that linked to this original sensory endowment is a reflex relationship with the iris, the cause of which connection is the more proper one to examine. It also reminds one of the intimate and very general connection between the irritation of a sensory nerve and the contraction of the muscles around the eye—the *orbiculares* and *corrugatores*. The evolution of the higher sensory endowments out of the more general substrata of sensation embraces yet in this remove these correlated movements, so that the emotional manifestations of their excitation include frequently the phenomena attendant upon simpler and more automatic conditions.

Pupillary Anomalies, &c., in Cerebro-spinal Diseases.—The

¹ 'The Expression of the Emotions in Man and Animals,' 1872, ch. xii. p. 304.

above considerations lead naturally to the query how far this extensive reflex relationship may be affected in morbid conditions of the brain and spinal cord; and with the object of replying to this question I carefully examined all the male epileptics and general paralytics in the West Riding Asylum; and in order that this examination should afford me a clue to the state of the sensory and motor strands of the spinal cord, it was thought essential to test the reflex apparatus by mechanical, thermal, and electric stimuli, to note for each case the presence, absence, or deficiency of normal sensory appreciation for such stimuli, and their effect on the dilatation of the pupil. My objects certainly were manifold, but one of them was to determine whether in the progress of those atrophic changes in the cord which histologists have pointed out in general paralysis, the loss of electro-muscular contractility keeps pace with the deterioration of reflex excitability and of sensory impressions and perceptions of touch, pain, heat, &c. The test by heat consisted in the sudden application to the sole of the foot of a spoon raised to the temperature of nearly 212° . Electro-muscular contractility was gauged by means of Stöhrer's double-celled induction battery, the secondary coil being invariably used, the hammer adjusted for a rapidly interrupted action, and the electrodes applied so as to transmit a direct current, the positive being nearer the centre than the negative electrode. The results are too voluminous for insertion in an article such as this, but, I have embraced in a tabular form their general features, having reduced the various items to the percentage form as the one more useful for a clear appreciation of the case. I will take the facts *seriatim*.

Loss of Reflex Excitability.—The more delicate appreciation for tickling was noticeably blunted in both general paralytics and epileptics, about 41 per cent. of the former and 21 per cent. of the latter being wholly unaffected, whilst a high percentage of both also exhibited great deficiency of reflex excitability and sluggish reaction to the same stimuli. To painful impressions, as of a prick with a needle, it was found that over 60 and 73 per cent. responded with normal vigour, but, that whilst in but 6 per cent. of the

Epileptics no movement ensued, at least 17 per cent. general paralytics showed not the *slightest* response (*vide* Table A). This more or less complete suspension of reflex manifestations in the lower limbs involves (leaving the condition of the muscle itself out of consideration) one of three elements either one or more of which may be at fault. The sensory, motor, or multipolar ganglionic apparatus of this reflex loop may be each in their turn implicated. What evidence have we obtained which leads us to infer which of the triple links in this excito-motor chain becomes chiefly affected? It will be noticed with regard to the General Paralytics that sensory appreciation, or more accurately, perception of *pain*, was more dulled than in Epileptics; still more palpably is this manifested with regard to their appreciation for *heat*—60 per cent. being wholly unaffected by a temperature of nearly 212°, and the remainder, by a large majority, speaking of it as ‘pleasant,’ ‘nice and warm.’ The Epileptics, however, who exhibited *no* response to heat were but *one-third* this number. The fibres, therefore, which conduct thermal impressions are deficient in their reflex relationships in the cord as well as their conductivity upwards to the sensorium commune.

TABLE A.—REFLEX EXCITABILITY AND ELECTRO-MUSCULAR CONTRACTILITY.

IN GENERAL PARALYSIS				IN EPILEPSY		
Stimulus	Nil	Sluggish	Active	Nil	Sluggish	Active
	p.c.	p.c.	p.c.	p.c.	p.c.	p.c.
Tickling . .	41·46	17·08	41·46	21·31	34·42	44·26
Pricking . .	17·08	21·95	60·97	6·55	19·67	73·77
Heat (212° F.)	67·97	12·19	26·82	21·31	26·22	52·44
Electricity . .	78·94	...	21·05	65·57	...	34·42

Reflex Dilatation of the Pupil.—But there is another trustworthy method for ascertaining the conductivity along the sensory columns of the cord. We cannot always trust to a patient’s assertion as to his appreciation of pain, and occasionally he is in no position to reply to our queries. In these cases we may always fall back upon the plan of

watching the pupils whilst the foot is pricked or otherwise irritated. Where the sensory columns are intact, such an impression is instantly signalled by the dilated pupil. In cases, therefore of cerebro-spinal disease, when this reflex dilatation of the pupil is active, we are in a position to affirm that the sensory strands of the cord as far as the cilio-spinal region are not seriously implicated. Table B. shows that about one half of either class of patients exhibited a deterioration of this reflex dilatability of the pupil, and of these in an average of 24 and 35 per cent. it was *absolutely* abolished. The greater interest of these facts, however, attaches itself to their comparison with other data. Thus as reflex movement of the legs is due to that segment of the cord only to which its nerves run, and is little or not at all affected by injuries at a higher level,¹ so we might infer that the absence of reflex dilatability of the pupil might be due to derangement of the sensory portion of this loop. Reference to the table, however, will at once undeceive us on this point, as it clearly showed that out of an average of 100 General Paralytics 18 showed a deranged action, and 20 absolute abolition, of this motor reflex action of the pupil, even with a *perfectly normal* and active reflex condition of the legs.

TABLE B.—REFLEX DILATATION OF PUPIL.

GENERAL PARALYSIS				EPILEPSY		
Reflex action in legs	Nil	Sluggish	Active	Nil	Sluggish	Active
	p.c.	p.c.	p.c.	p.c.	p.c.	p.c.
Nil	7·69	2·56	7·69	6·89	∞	∞
Sluggish . .	7·69	7·69	5·12	3·44	8·62	8·62
Active . . .	20·48	18·04	23·04	13·78	15·5	43·10
Totals . .	35·89	28·22	35·89	24·08	24·08	51·72

This evidently points to a lesion at a higher level than the lumbar enlargement cutting off communication of the lower limbs with the cilio-spinal region of the cord. This

¹ I must here make exception of those cases of destructive lesions of the thalamus opticus which, as Dr. Crichton Browne has pointed out, are invariably accompanied by loss of reflex spinal action. *Vide* 'West Riding Asylum Reports,' vol. v.

implication of the higher spinal tracts whilst lower dorsal and lumbar segments remained functionally intact, was a characteristic feature in my examination of both General Paralytics and Epileptics, occurring in either class with about the same relative frequency.

The interference with the normal reflex dilatation of the pupil was in several cases partial only, and in a few cases it was *unilateral*, and if associated with deficient reflex activity of the limbs or of one limb, the pupil affected was that *opposite* to the member chiefly implicated. All these cases were carefully tested with atropine which caused marked dilatation. It has been asserted by not a few writers, that the earlier stages of general paralysis are almost invariably marked by a contracted pupil coincident with the congestive conditions preceding the atrophic changes which are ushered in at a later period. The latter stages of general paralysis, according to these authors, show us usually a dilated pupil corresponding to the period of atrophy.

Griesinger distinctly states that, 'at the commencement *the pupils* are often regularly contracted; afterwards they again enlarge, but often unequally.'¹ My own experience, confirmed by the views of one at least of my colleagues here, tends rather to prove that the *contracted* pupil is a frequent accompaniment of the very latest stage of the disease. As facts are worth a bushel of opinions, I subjoin a table in which the duration of the disease in 40 general paralytics is stated, together with careful measurements of both pupils, and it will, I think, be observed that the list given by no means favours the assumption referred to above.²

¹ Griesinger on 'Mental Diseases,' New Sydenham Society, 1867, p. 396.

² It is a noteworthy fact that many advanced general paralytics can read clearly even the smaller text types of Jäeger. This, however, need not surprise us when we remember how perfectly sight has been maintained even in acute forms of optic neuritis, a point indeed which Dr. Hughlings Jackson has frequently insisted upon.

TABLE C.—STATE OF PUPILS IN 40 GENERAL PARALYTICS.

Names	Supposed duration of disease	Measurement of pupils in millimetres	
		1·5 right	1·5 left
1. J. O.	1 month	2	2
2. T. H.	2 months	2	2
3. J. McG.	6 "	2	1·5
4. W. B.	6 "	2·5	2
5. D. D.	6 "	3	3
6. W. B.	6 "	4	3·5
7. T. S.	6 "	3	3·5
8. A. H.	6 "	3	3·5
9. M. H.	6 "	3·5	4·5
10. M. S.	From 6 months to 12 months	2	2
11. W. W.	" "	3	3
12. W. B.	" "	1	1
13. M. S.	Above 1 year but under 2 years	1	1
14. G. M.	" "	1	1·5
15. E. C.	" "	3	3
16. T. G.	" "	3	3·2
17. T. M.	" "	3·5	3·5
18. W. A.	" "	2	2
19. J. D.	" "	4	1
20. J. M.	" "	2·5	2·5
21. R. F.	Above 2 years, but under 3 years	2	2
22. W. S.	" "	3	3
23. G. P.	" "	2·5	3
24. J. P.	" "	2·5	2·5
25. W. W.	" "	5	2
26. T. G.	" "	4	4
27. J. E.	" "	3·5	4
28. A. S.	Of 3 years standing	2·5	2·5
29. T. B.	" "	3	3
30. E. G.	" "	3	3
31. D. M.	Of 4 years standing	1·5	1·5
32. T. L.	" "	1·5	1·5
33. J. D.	" "	2·5	2·5
34. R. S.	" "	4·5	4·5
35. B. A.	Of 5 years standing	1	1·25
36. C. W.	Of 6 "	1·5	1·5
37. H. Sl.	Of several years (uncertain)	2	1
38. J. N.	" "	1	2
39. H. E.	" "	Cataract	...
40. G. N.	" "	2·5 right	2·5 "

Inequality of Pupils.—The inequality of the pupils in cerebral disease is a symptom universally acknowledged as carrying with it in most cases a very serious prognostic indication. My object in referring to it here is to attach to it a greater weight in the *topographical* diagnosis of cerebral lesions. That it bears such an indication I feel strongly convinced, my views being based entirely upon clinical data;

in fact, I regard it in most cases as of extreme value as a *localising* symptom. In cases of mental disease, of epilepsy and other discharging lesions induced by *coarse* disease and accompanied by marked inequality of the pupils, I believe the unequal pupils serve to localise the lesion hemispherically when we are able to eliminate such fallacies as direct pressure upon certain nerve tracts from tumour, or the presence of intra-ocular disease. Cases of general paralysis, as is well known, afford us a large majority of unequal pupils. Thus Seifert ('*Zeitschr. f. Psych.*', x. p. 561), quoted by Griesinger,¹ observed 17 cases in 25 paralytics. I have not, however, confined myself to General Paralytics in investigating this question, but, draw my conclusions from the history of Akinesia and Hyperkinesia generally. The table of these results exhibits epileptics, general paralytics, cases of chronic atrophy, dementers from other causes &c., in whom discharging or destroying lesions were localised in one or other of the hemispheres; thus affording us a clue to the localising value of a prior fixed irregularity in the pupils. I believe we may find in these results strong grounds for belief that the *dilated pupil is on the side of the lesion*, and thus if we note in a general paralytic that the left pupil is the larger of the two,² and the patient be seized with a congestive or epileptiform attack, the chances are that he will suffer from *right* hemiplegia or convulsions, *beginning* if not *confined entirely* to the right-half of the body. It is of importance also to note that during and after the convulsion or paralysis the condition of the pupils may be quite reversed, as I have had frequent opportunities of verifying. As one instance I may quote a case occurring to me only the day prior to my writing these lines, in which a general paralytic was seized with unilateral convulsive twitchings of the face and limbs limited to the *right side*; the *right pupil* was the larger. On reference to my notes, however, I found he had for at least a month or more a decided dilatation of the *left* pupil, the right being much smaller. In two hours time, the

¹ Op. cit. p. 396.

² Eliminating of course such fallacies as intra-ocular changes, &c.

convulsions having ceased, the left had again become the larger of the two and remains so still.

TABLE D.—ILLUSTRATIVE OF PUPILARY ANOMALIES IN CEREBRAL LESIONS.

Number of case	State of pupils <i>prior</i> to seizure	Nature and site of seizure
1	Left pupil largest	Convulsions of right half of body
2	" " "	Right hemiplegia.
3	" " "	" "
4	" " "	" "
5	Right " "	{ Convulsions, and then paralysis of left half of body.
		{ Right hemiplegia with aphasia.
6	Left " "	{ Repeated convulsive seizures, always of right side, and the head and eyes drawn to the same side.
		{ Frequent tonic convulsions of right side of face, the head drawn to right side.
7	" " "	{ Right hemiplegia.
8	" " "	{ Convulsions, head and eyes always to the right side.
9	" " "	{ Convulsions, and then paralysis of left side.
10	Right " "	{ Right hemiplegia with aphasia.
11	Left " "	{ Repeated convulsions, always affecting the <i>right</i> side only.
12	" " "	Convulsions of left side.
13	Right " "	" right side.
14	Left " "	Left hemiplegia.
15	Right " "	Right hemiplegia.
16	Left " "	Left side convulsed and paralysed.
17	Right " "	Left hemiplegia.
18	" " "	Slight paralysis of left leg.
19	" " "	Right side convulsed.
20	Left " "	Convulsions of right side.
21	" " "	" "
22	Right " "	" "
23	Right largest after seizure	Right hemiplegia.
24	Left " "	Convulsions of left side.
25	Right " "	" right side.
26	" " "	Right hemiplegia.

Observations.—The above table was compiled from three volumes of the Asylum Case Books, and might have been greatly extended had space permitted. All cases were included where markedly irregular pupils were *succeeded* by paralytic or convulsive seizures. It will be observed that Case 22 is the only exception to the rule already stated; as in Cases 23 to 26 the observation of the pupil was made only

after the seizure, which almost invariably alters the previous condition of the pupils, as regards relative size, temporarily, the smaller often becoming the larger pupil for some time after the convulsive affection has subsided.

Electro-muscular Contractility.—It is interesting to note the difference exhibited in general paralytics, as to susceptibility to the reflex stimulation of mechanical, thermal, and electric agents, so that where the pricking of a sharp needle produced no effect in but 17 per cent., heat and electric shock were unavailing in 60 and 78 per cent. respectively. It is stated by Griesinger that ‘the contractility of the muscles under the influence of electricity is, as in other forms of cerebral paralysis, always normal.’ His conclusions on this point are in direct antagonism to my own, but, repeated observations have fully confirmed my opinion that electro-muscular contractility is markedly and decidedly diminished.

In this opinion, however, I am supported by no less an authority than Dr. Bucknill, who published the result of his enquiries on this point many years ago. On reference to the table it will be observed that a percentage of 78·94 general paralytics showed no response, or but the slightest fibrillar tremor on applying a centripetal current (direct) from the secondary coil of a Stöhrer’s battery, the gauge being drawn up to zero.

VII.—ON QUASI-CONVULSIVE MOVEMENTS IN EPILEPSY.

In connection with apparent correlations existing between the resultants of sensori-motor excitation, or in other words betwixt motor and thermal discharges occurring in certain convulsive affections, I think we may assume the occasional existence in epileptics of an unnatural accumulation of potential energy which may manifest itself by *slight*, though *constant*, overflow, either as motor or thermal phenomena, and which perhaps may serve to postpone a convulsive attack, as it is only by the *excessive* storing up of energy that a general epileptic discharge would be induced. An analogous condition to that which I allude to is exhibited in the states which we term the ‘fidgets,’ and which Sir H. Holland refers

to as 'an accumulation of some cause of irritation which requires muscular action for its relief.'¹

Amongst the numerous cases of epileptics at present in the West Riding Asylum, there are at least three which typically represent what I here allude to. In these cases the convulsive seizures are of a very violent and general kind, in one subject followed invariably by an outburst of the wildest maniacal excitement, in another by intense physical and mental prostration together with amaurosis. The inter-paroxysmal period is always marked by a constant fidgety movement of the fingers and hands. The movements vary from the most simple to the most complex. More commonly there is rapid extension of the fingers, which are spread widely apart, and then ensues a slower flexion, first of the ungual series of phalanges, then the middle, and lastly the metacarpal group, when the fist is clenched with much vigour. These movements are repeated over and over again, but are frequently complicated by adduction and abduction of the fingers and rotatory movements of the thumb. The patients can arrest these movements volitionally when directed to do so, and appear conscious of their awkward and incongruous nature, as, when they perceive one's attention directed to them, they frequently expend these movements in some apparently purposive actions, as in buttoning and unbuttoning their coats. There is decided deficiency of cutaneous sensibility over both hands, and the patients state that the fingers become 'more and more stiff in feeling.' The feet do not appear to be involved in like manner.

These movements must not be confounded with that group of symptoms which Hammond regards as a special affection *sui generis*, and which has been dignified by the title of athetosis, inasmuch as they are not of so limited a character, and are in all of his three cases bi-lateral. The feet are not involved in like manner.

¹ Quoted Darwin in his 'Expression of the Emotions,' 1872.

VIII.—THE ECSTASY IN ACUTE DEMENTIA.

The following notes taken of a case admitted into the Asylum, as an acute dement, may prove of interest. The lad had shown after admission no very acute symptoms, and had latterly been employed in the service of the butcher of the establishment, when, with no premonitory warning, the following attack supervened.

August 30.—To-day, when entering the Asylum with the butcher, he threw himself deliberately before the cart-wheels with suicidal intentions. He, however, was saved, and escaped with but a few bruises, and on being examined it was found that he had become obstinately silent and sullen, and exhibited also such symptoms of prostration as to necessitate his being put to bed.

August 31.—This morning he got out of bed on several occasions, and tried to injure himself by running his head against the wall of the room. He was placed under special charge.

September 1.—Lies in bed obstinately silent, his eyes widely staring, the pupils dilated to the extent of 8·5 millimetres, yet contracting readily to light. His face is expressionless, vacant, and the mouth partially open, but there is no dribbling of saliva. Extremities bluish and cold. Cheeks flushed. When taken out of bed, he clings to the attendant or falls in the most helpless manner to the ground. Takes his food when fed with the spoon.

September 3.—Remains in the same apathetic trance-like condition. Has to be fed by the funnel, and on these occasions he has once or twice manifested his appreciation of his surroundings by offering to swallow his food, so as to avoid being forcibly fed. He also attends to the calls of nature, but speedily relapses into the same mental state. His pupils still measure 8 millimetres; his face greatly flushed. Temperature in axilla, 96·8° F.; pulse, 74; breathing 24, very shallow. Arms remain fixed rigidly in whatever position they are placed. This cataleptiform state has existed for two days. Not the slightest movement of the facial muscles is observed; even the blinking of the eyelids is suspended.

September 4.—Temperature in axilla, 97·8° F.; pulse 64; respirations 20. Still refuses food. Reflex action almost abolished in both soles to tickling, pricking, or heat. In regard to electro-muscular excitability, the extensors and flexors of the toes are just brought into action by a current of Stöhrer's double-celled battery from the secondary coil, the gauge being drawn up to 10. The pupils are in a peculiar oscillating condition, dilating and contracting almost rhythmically. They also contract to light, but dilate to the fullest extent afterwards. Cataleptic condition still persists. The limbs are cold and pale.—Later in the day the temperature fell to 96·1°, the pulse to 60, the respirations being 22. The pupils lost their oscillating movements, became dilated to the *fullest* extent, were equal and quite insensible

to the strong glare from a bull's-eye lantern. Cheeks still almost scarlet in hue; conjunctivæ injected. Respiratory murmur too feeble to be heard.

September 5.—To the above symptoms are added the occurrence of dribbling of saliva. Temperature at night, 97·1° F.

September 6.—In a state of profound mental torpor; he never speaks, and is apparently utterly indifferent to all around him. Reflex action totally abolished. The cataleptic phenomena have subsided. The other symptoms remain *in statu quo*. Nitrite of amyl inhalations cause a slight quivering of the eye-brows and lower lip, with movements similar to those of deglutition. The respirations deepen somewhat in character; there is also copious lachrymation, and a breaking out of a profuse perspiration over the surface of the face. Temperature 97·6° F.; pulse 84; respirations 16. There is no reflex excitability in the limbs, but the muscles of the face respond readily to the stimulus of pricking. The muscles of the thorax respond normally on percussion.

September 8.—The legs have become quite warm again. The eyelids droop naturally, and the pupils measure 4·5 millimetres. The eyes water freely, and the eyelids blink to the stimulus of light, and on passing the hand across the face. Temperature 97·8° F.; pulse 78; respirations 14.

September 12.—The general symptoms have much improved, but he still requires the forcible administration of food. The temperature has risen to 99·1° F., but fell towards evening to 98·3° F.

The case just cited serves well to illustrate the apparent connection existing between acute or primary dementia and melancholia attonita, and the occurrence of such cases is ample excuse for those who have erred by regarding these two terms as synonymous, rather than as embracing two distinct entities with a clear differential symptomatology attached to each. The onset here was that of a sudden, profound melancholy, the self-consciousness wrapt in an all-absorbing painful emotion, fettering the will to its morbid behests, resulting in determined and repeated suicidal attempts. Thence the passage into a condition of passive indifference, increasing callousness to objective impressions, combined with those peculiar cataleptic states which so frequently present themselves in similar cases. In this stage, however, it will be observed that the patient still retained a certain evident appreciation of his surroundings, the expression was *distressed*, and the case might very fairly have been regarded as one of atonic melancholia. Lastly, we find him falling into a still more profound stupor, a condition approaching the extremes of self-abstraction, an apparent utter abrogation of the will with complete intellectual torpor.

The dilatation of the pupil unaffected by the stimulus of a strong glare of light gave way on two occasions to the peculiar rhythmic oscillation referred to in the case. This condition I have previously observed in a case of epilepsy during recovery from the 'status,' and it is probably attributable to vaso-motor disturbances. The latter stage assumed the usual features of acute dementia as far as the expressionless face, general anæsthesia, and vaso-motor derangements were concerned. It is noticeable, however, that the obstinate vomiting and profuse salivation which are so often met with were absent here, nor did the habits of the patient become to any degree dirty and degraded. It is interesting also to note that whilst the limbs were pale and frigid the face was much flushed, being of a bright arterial hue, the conjunctivæ injected, and this vascular dilatation appeared peculiarly limited to the area of distribution of the trifacial nerve. In view of the strong arguments adduced by Dr. Crichton Browne, as to the suspension of reflex irritability in lesions of the thalami optici, and regarding these ganglia as the probable seats of those complex integrations which we term the sensational and perceptive faculties; cognisant as we also are of its probable close relationship to a vaso-motor centre, we might be led to suspect these regions as being especially involved in cases such as the one we have been considering. More especially does this remark apply to those earlier stages of the affection marked by the cataleptic seizures, the reduction to an automatic vegetative existence; but prior to that obliteration of memory and psychical oblivion which necessarily constitute the essential elements of extreme dementia. We have learnt to recognise in cases of extreme atonic melancholia, that whilst the sensorial perceptions may be wholly reactionless to the stimuli supplied by the environment, yet they may be keenly alive to an *inner life* of which the patients on recovery make the strangest revelations, a predominance of that pain accompanies the perceptive faculties which is closely connected with what Griesinger has termed the limitation and degradation of the Ego. I think it probable that (as this case tends to prove) such a state may precede the development of acute dementia.

THE CRANIAL OUTLINE OF THE INSANE AND CRIMINAL.

By CROCHLEY CLAPHAM, L.R.C.P. LOND.,

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AND

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THE shape and size of the head have always been considered as bearing a relation to the degree of intelligence of the individual, and many an estimate of mental capacity has been made from the mere expansiveness or otherwise of the forehead. What, for instance, is a more common expression than 'So and so must be an exceedingly intelligent man; see what a noble forehead he has!' And although we may deny that any such prejudice holds a place in our own estimate of character, yet are we nevertheless possessed by a certain, perhaps unconscious, substratum of reverence for the possessor of a 'fine head.' Consequent upon this veneration of the skull capacious, has arisen an unreasoning respect for heavy brains, and, unless in the case of a secluded lunatic, the weight of a man's brain has been used as a measure of his wit. That such a theory is incorrect, not all the instances of great men with small heads have been sufficient to prove to the satisfaction of the Megacephalophiles. And although all the other organs of a healthy body are

deemed capable of sufficient activity when called upon to show it, notwithstanding their ordinary size, yet is the brain singled out to be worshipped, like a Hindoo god, in proportion to its bulk.

If one ventures to suggest the possibility of great value attaching to a small coin, the brains of Cuvier, Gauss, and a dozen others are referred to, with the inference that Gauss, and Cuvier, would have been nothing in particular had their brains been a couple of ounces lighter. Their genius being associated with, is supposed to result from, their large brain-weight, but, we take it, the 'propter' is hardly proved. It is forgotten that the fact of great genius having co-existed in one single instance with a small head is sufficient to entirely upset this theory. And should 48 ounces of brain suffice to contain and elaborate the wit and wisdom of a great philosopher—and who will contend that such has not been the case?—what need of further weight? ¹

In such a strain Wagner spoke, and opposed the conclusion that weight of brain meant wealth of wit. So also Barnard Davis—than whom no greater authority on crania exists—writes ² on this subject: 'My observations agree with those of Wagner, that weight of brain does not indicate any close relation to intellectual power, and also that aboriginal races are not to be distinguished for smallness of brains. In fact the Ancient Britons, and I may add the Ancient Gauls also, were remarkable for good-sized, nay even large brains.'

The above expression of opinion was drawn from Dr. Davis by the perusal of a paper, on the weight of the brains of some Pelew Islanders and Chinese, which was forwarded to him previous to its being submitted to the Anthropological Institute. It was there shown that, in a number of uneducated, ill-regulated, but muscular men, the brain average was considerably above that of Europeans generally, and it was ventured to draw the provisional conclusion that the excess of brain matter was musculo-motor, not mental in function. But for particulars of brain weight the reader is referred to page 11 of this volume, the present paper being

¹ The late Professor Laycock's brain weighed only 48 ounces.

² In a letter to one of the authors.

devoted to the consideration of the external size and configuration of the skull.

Attention having been directed by Dr. Crichton Browne to an instrument called a 'conformateur,' used by hatters to obtain the form of the head, and to the possible value of its application to the crania of the insane as a means of discovering their prevailing shape, the present series of observations were instituted. We were enabled by the courtesy of Mr. Carruthers, of Wakefield, to whom we are indebted for the loan of the 'conformateur,' to carry out observations upon the heads of nearly 1,300 patients, male and female, in the West Riding Asylum, of 500 criminals in the West Riding Prison, and of a number of sane individuals of both sexes outside these institutions.

We commenced our examination in the full persuasion that we should meet with a remarkable want of symmetry in the skull-shapes of the insane, but were much astonished to find that the generality presented nothing essentially peculiar in their configuration, and that, in fact, their skulls were not nearly so twisted as their wits.

The general impression is, we believe, that the heads of the insane are markedly asymmetrical, and even Dr. Wilks, in addressing one of us on this subject, says: 'I have purchased a number of photographs taken from prisoners in gaol, and there is scarcely a decent head amongst them, *they might well be taken for inmates of an asylum.*' This is a libel upon the insane generally, and, coming as it does from one who is well known to take a lively interest in everything connected with insanity, sufficiently shows what is the prevailing impression as to the insane skull-shape even amongst scientific men.

On this point we are convinced from our own observations that the heads in this Asylum will compare very favourably, measured by the present standard of excellence, with the general average of sane persons, either as regards capacity, regional development, or symmetry.

That there are asymmetrical and distorted heads amongst the insane is undoubted, but the same is true of the sane; and with the exception of one type of head, which, so far as

we can at present judge, is confined to the insane, we are able to match all the shapes observable in the insane with examples from the heads of sane people. The exception of which we speak, and which we have named the *insane type*, is the skull-shape in which the greatest transverse diameter is placed in the anterior third of the skull, and is in fact the head possessed of the noble forehead of which so much is at present expected.

It may be that this is only another proof of how near akin great wits and madmen are; but, be that as it may, it at least also shows that such a frontal development is not a possession so ardently to be desired as we have hitherto been taught to believe.

Speaking generally, the outlines met with may be divided into three classes, having the greatest transverse diameter in the anterior, middle, and posterior thirds of the skull respectively.

The first class was composed solely of lunatics, no instance having yet been met with amongst sane people; indeed, in those of sound mind it is an occurrence of great rarity for the greatest transverse diameter to be found anterior to the central point of the skull.

When studying this point we were much struck with the deceptive appearance of foreheads when viewed from the front, and it was not until we adopted Lavater's stand-point, and examined them from above, that we were able to determine their true value as regards capaciousness. The removal of the hair from the temples has the effect of greatly increasing the apparent breadth of foreheads which, although coming almost to a point as seen when viewed from above, may on a front view being taken of them, by virtue of foreshortening, present a broad and expansive appearance.

Of the insane 7.75 per cent. belonged to the first class, and the great majority of these were general paralytics and epileptics, as will be seen on referring to the table of regional development.

How much of this frontal preponderance is due to increased development of this part, and how much is due to diminution of the capacity of the rest of the skull will not

be considered in the present paper. That the skull does alter in size to a very marked extent was conclusively shown by Dr. Yellowlees in his Report of the case of Smith, the Morningside Murderer,¹ and there is little doubt that some of the variations in this direction may be explicable on similar grounds.

To the second class, however, belonged the great majority of the insane, 82·275 per cent. of them having the greatest transverse diameter in the middle third.

Of the third class there were 9·975 per cent. of the insane.

The anterior half of the skull was the seat of the greatest transverse diameter in 21·95 per cent. of the insane, and the posterior half of 78·05 per cent., and in criminals 2 per cent., and 98 per cent. respectively.

From the foregoing it will be seen that the most common form of skull met with was that having its greatest transverse diameter posterior to the median transverse line, and being most protuberant, with reference to the long diameter, on the left side. This condition of left-headedness was present in 81·771 per cent. of the insane; but, as shown in the regional development table, was much more marked in some diseases than in others, and as bearing upon the question of which is the 'driving-side' for convulsions, it may be interesting to notice that left-headedness is not only very common in epilepsy, but is also more pronounced in this disease than in any other form of mental affection. This seems to accord with Dr. Hughlings Jackson's theory of the left side being especially motor in function.

The most symmetrical heads were those of imbeciles, of which 25 per cent. had this character.

The numerical superiority of the 'left headed' agrees with observations made by numerous writers on the greater relative weight of the left cerebral hemisphere. That it has an obvious, though by no means exclusive, connection with right-handedness is shown on comparing the criminal tables where the opposite state of right-headedness was exhibited in a number of left-handed individuals.

¹ 'Edinburgh Medical Journal,' August, 1862.

The hand employed in throwing a stone was the test used to determine the right or left-handedness of the individual. But, as will be easily understood, this test could scarcely be applied to lunatics, and their personal depositions were not considered sufficiently reliable for scientific purposes. The hand used in cutting the food was thought open to objection as a test, from the fact that many children who are naturally, and in every other way persistently, left-handed, are accustomed by their parents under pain of chastisement to take the knife in the right hand. Stones, however, are usually thrown in the absence of the parent, and any punishment meted out as a corrective of this bad habit is usually independent of the hand used in their projection.

Left-headedness was much more marked in men than in women, and this goes to substantiate the 'crossed-action' theory, as most female employments necessitate the pretty-even use of the two hands; those which are exclusively one-handed being generally light and not requiring much 'driving-power.'

Amongst the criminals who were left-handed the posterior half of the head was larger on the *right* side in 50 per cent. on the left side in 30 per cent., and equal on the two sides in 20 per cent.

The right half of the skull was almost invariably in advance of the left half, the whole side appearing to be pushed bodily forwards, and both its frontal and occipital limits placed anterior to those of the opposite side.

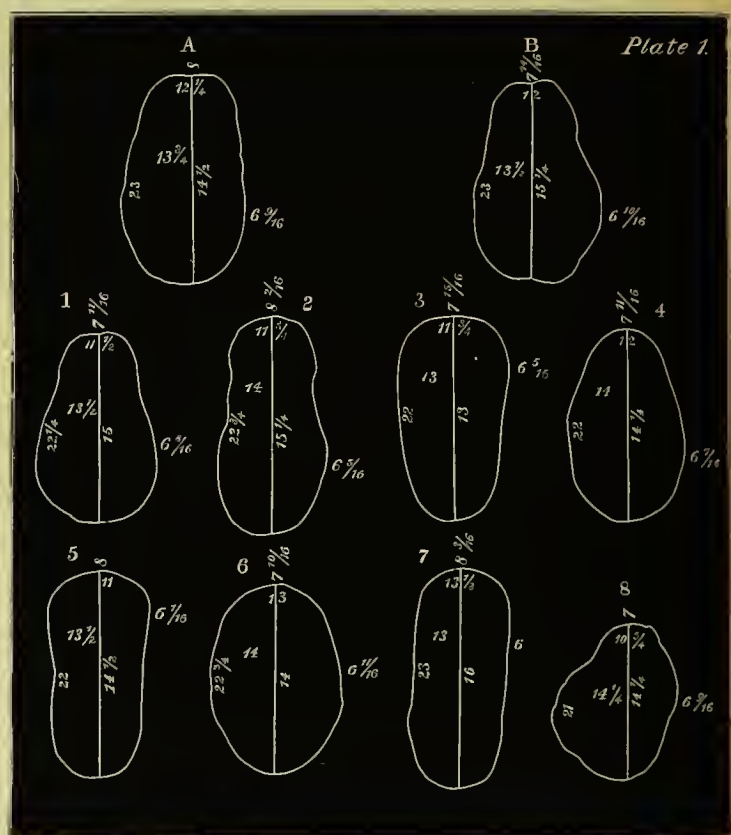
The above facts as far as they relate to one-sidedness are true of the level at which these outlines were taken (*vide infra*), and from observations taken in some few cases we have reason to believe that the above features become even more declared in sections of the skull taken at a lower level, and less so as the point of section is raised.

The principal shapes met with may be conveniently grouped as follows—the terms used being adopted from botanical nomenclature, and sufficiently explaining themselves—the nose being taken as representing the position of the stalk.

- (1.) Pyriform.
- (2.) Panduriform.
- (3.) Ovate.
- (4.) Obovate.

- (5.) Cuneiform.¹
- (6.) Rotund.
- (7.) Oblong.

NOTE.—The examples of these outlines obtained by the use of the 'Conformateur,' and given in the accompanying Plate (I.), being reduced ovoids, must not be taken as representing the actual shape of the head. They are used only as a means of classification and comparison, their points of diversity coming out much more strongly in the reduced than in the normal size.²



¹ No. 5 is the exclusively insane shape.

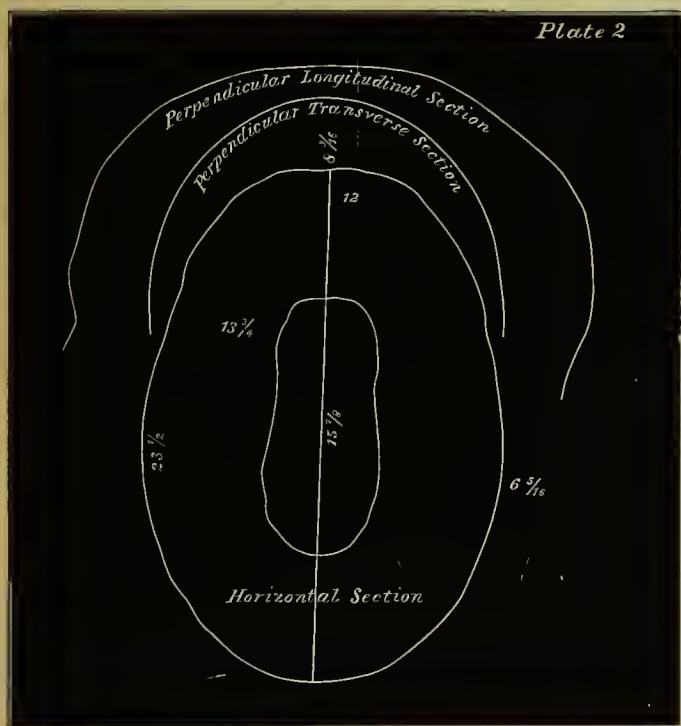
² Enlarged shape shown in plate II.

In dealing with the details the insane and criminal are considered as much as possible together so as to facilitate comparison.

The cranial outlines were taken just above the level of the supraorbital ridge and external occipital protuberance, and represent a horizontal section of the skull in this plane.

Whole circumference was measured above supraorbital ridge and over occipital protuberance.

Frontal circumference round forehead above supraorbital ridges from a point above one auditory meatus, to a point above the other on the circumferential line.



Antero-posterior arch from external occipital protuberance to root of nose. Transverse arch from auditory meatus to auditory meatus over vault.

The diameters, antero-posterior and transverse, were respectively the longest contained in the skull.

The subject matter is arranged under the following heads :—Size, Age, Height, Weight, Complexion, Disease, Sex, Nationality, Right and Left-Handedness, Sane and Insane.

SIZE.

The average size of the head was considerably larger in the insane than in the criminals, notwithstanding the idiots included in the former class.

Of the 629 insane males examined 20·349 per cent., had heads of 23 inches and upwards in circumference, and these also showed a corresponding increase in the other dimensions of the skull with the exception of the transverse arch, which maintained much the same length in all the averages—large heads, small heads, and idiots. This, which is a very marked feature, is, no doubt due to the early closure of the parietal portion of the sagittal suture. The subjoined table exhibits this peculiarity.

	Av. age	Av. height	Circumference		Arch		Diameter	
			Whole	Frontal	Ant. post.	Transverse	Ant. post.	Transverse
Of 23 ins. and over	45·917	65·630	23·225	12·149	14·798	13·986	8·029	6·464
Idiots	23·210	57·625	21·684	11·342	13·769	13·343	7·434	6·161
Difference . .	22·707	8·005	1·541	·807	1·029	·638	·595	·303

Of the criminals only 6·4 per cent. had heads of 23 inches and upwards in circumference.

SEX.

Male heads were much larger than female, the average of the latter, indeed, being in all their dimensions below that of male idiocy. This fact alone is, we think, sufficient to show the non-essential character of size. The proportions in the two sexes were as follows :

Sex	Age	Height	Circumference		Arch		Diameter	
			Whole	Frontal	Ant. post.	Transverse	Ant. post.	Transverse
Male . . .	43·589	65·114	22·205	11·663	14·182	13·484	7·738	6·253
Female	21·301	11·045	13·277	12·980
Difference	·904	·618	·905	·504

The age, height, and head diameters of the females have not yet been taken, and the comparison is made in its present rudimentary form for what it is worth, a further and much more elaborate treatment of the whole subject in all its bearings being contemplated by the authors, at an early date, to which the present paper is only preliminary.

Comparing these with the measurements made in a number of cases in the Somerset County Asylum by Dr. Boyd, we find that the average circumference in both males and females was the same as our own, but that in the case of males our average antero-posterior arch was ·982 inch and our transverse arch ·284 in. longer than his, and in the females ·477 in., and ·381 in. longer respectively.

The rest of the paper treats exclusively of males.

The general average measurements of insane and criminals, were as under.

Class	Age	Height	Circumference		Arch		Diameter	
			Whole	Frontal	Ant. post.	Transverse	Ant. post.	Transverse
Insane . . .	43·589	65·114	22·205	11·663	14·182	13·484	7·738	6·252
Criminal . .	30·698	64·612	22·015	10·702	14·074	13·327	7·612	6·223
Difference .	12·891	·502	·190	·961	·108	·157	·126	·029

Here the most marked difference is in the frontal measurement.

Of the criminals 6 per cent. were left-handed, and the peculiarities of their cranial outline may be gathered from the following table.

	Larger on right side behind	Larger on left side behind	Larger on right side in front	Larger on left side in front	Equal behind	Equal in front
	p.c.	p.c.	p.c.	p.c.	p.c.	p.c.
Right-handed . .	22·46	49·70	59·32	10·38	27·75	30·29
Left-handed . .	50·00	30·00	40·00	36·66	20·00	23·33

It appears, therefore, from an examination of these figures, that amongst right-handed men it is more usual to have the left side posteriorly larger than the right, and amongst left-handed men the right side larger than the left, while the right side anteriorly is more commonly larger than the left in both, but to a much less extent amongst left-handed people.

There is commonly a considerable flattening of the posterior cranial curve on the right side, which may also be seen sometimes, but to a less extent, on the left. This last was not nearly so well marked in the insane as in the criminals.

Dr. Merei, as quoted by Professor Humphry in his work on the 'Human Skeleton' (p. 189, foot-note), says: 'In the majority of cases, according to my experience, I may say small skulls are sooner completely ossified than large ones, but there are numerous exceptions to this rule.' In connection with this expression of opinion it is interesting to find that our small skulls are remarkable for symmetry, which, indeed, is the most striking point about them, whilst large heads are, with the exception of hydrocephalic ones, inclined to be one-sided or otherwise asymmetrical. The average length of the European skull is given by Dr. Carpenter as 7·04 in., and its breadth as 5·47 in.; which measurements, we presume, were made on the dry bone. Allowing half-an-inch for the integuments included in our own measurements, our averages for the insane are still considerably above those of Dr. Carpenter, although our measurements of criminals agree fairly well with his estimate.

Outlines of the vault in the line of the two arches have been taken by us in a number of cases with some interesting results, but particulars of these are held over until our next paper. We may premise, however, that our observations in this direction tend to confirm the opinion that dolichocephala-

lism, the result of early sutural synostosis, is accompanied by marked occipital prominence.

AGE.

In both criminals and lunatics the skull showed a larger average size for those above 40 years of age as compared with the average of those under 40 years.

Class	Age	Height	Circumference		Arch		Diameter	
			Whole	Frontal	Ant. post.	Trans- verse	Ant. post.	Trans- verse
<i>Insane.</i>								
Above 40 . .	53·2	65·365	22·268	11·683	14·134	13·442	7·734	6·273
Under 40 . .	30·242	64·741	22·114	11·634	14·252	13·551	7·744	6·222
Difference .	+ 22·958	+ ·624	+ ·154	+ ·049	- ·118	- ·109	- ·010	+ ·051
<i>Criminals.</i>								
Above 40 . .	48·287	65·650	22·044	10·748	14·086	13·244	7·582	6·201
Under 40 . .	25·853	64·500	21·941	10·677	14·046	13·310	7·546	6·198
Difference .	+ 22·434	+ 1·150	+ ·103	+ ·071	+ ·040	- ·066	+ ·036	+ ·003

In lunatics above 40 years of age the height, circumference, whole and frontal, and the transverse diameter were greater, but both arches and the antero-posterior diameter were shorter. In the criminals the transverse arch alone was shorter in those above 40 years of age, the other measurements being greater than in the younger men.

It is possible that this apparent shortening of the arches may be due to the fact of those over 40 having a less quantity of hair than their younger brethren, but the shortening of the antero-posterior diameter is not explicable on these grounds.

The criminals were further divided as regards age into decades, their ages varying from 15 to 70 years. The figures, it will be noticed, gradually augment for the first four periods, so that there seems to be little doubt that the head does not reach its full development until at least 40 years of age; or perhaps it would be safer to say that it is capable of increase in size up to this age, though it is very questionable

whether, under ordinary circumstances, it undergoes any further enlargement.

Age	Circumference		Arch		Diameter	
	Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
Under 20 .	21·700	10·477	13·938	13·229	7·397	6·127
20 to 30 .	21·992	10·791	14·022	13·366	7·589	6·316
30 to 40 .	22·132	10·764	14·178	13·334	7·651	6·150
40 to 50 .	22·172	10·797	14·123	13·306	7·674	6·263
50 to 60 .	21·822	10·661	13·911	13·072	7·541	6·105
60 to 70 .	22·137	10·787	14·225	13·350	7·531	6·237

A good index is obtained for estimating the relative value of skulls by adding together the whole circumference and the two arches. This *cranial index* includes the measurements which are of most importance in relation to skull capacity. On applying this index to the above table we find that it reaches its highest value in the decennial period from 30 to 40, leaving out of consideration the last period, in which there were too few cases to be of much value.

COMPLEXION.

The chief point noticed with regard to the complexion was that, in both Criminals and Lunatics, the size of the head increased for dark people and decreased for light.

Insane.

Complexion	Age	Height	Circumference		Arch		Diameter	
			Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
<i>Insane.</i>								
Dark . . .	43·150	65·167	22·229	11·666	14·271	13·492	7·751	6·261
Fair . . .	44·446	65·013	22·158	11·657	14·008	13·470	7·712	6·236
Difference .	-1·296	+·154	+·071	+·009	+·263	+·022	+·039	+·025
<i>Criminals.</i>								
Dark . . .	32·132	64·670	22·077	10·697	14·183	13·408	7·545	6·258
Fair . . .	29·264	63·874	21·977	10·673	14·034	13·313	7·605	6·213
Difference .	+2·868	+·796	+0·100	+0·024	+0·149	+0·095	-0·060	+0·045

The Criminals were further differentiated as under.

Criminals.

Hair	Eyes	Circumference		Arch		Diameter	
		Whole	Frontal	Ant.-post.	Transverse	Ant.-post	Transverse
Brown	Blue	22.136	10.815	14.113	13.363	7.714	6.171
	Brown	21.929	10.666	14.068	13.386	7.553	6.228
	Grey	21.975	10.648	14.022	13.294	7.608	6.215
	Hazel	21.975	10.765	14.053	13.316	7.620	6.198
Dark Brown	Blue	22.000	10.750	14.187	13.437	7.562	6.156
	Brown	22.136	10.757	14.218	13.496	7.441	6.285
	Grey	22.025	10.610	14.210	13.395	7.592	6.263
	Hazel	22.000	10.740	13.961	13.269	7.586	6.171
Black	Brown	21.916	10.625	14.166	13.312	7.635	6.239
	Grey	22.500	10.562	14.625	13.125	7.937	6.281
	Hazel	22.750	11.000	14.500	13.875	7.812	6.625
<i>General Averages.</i>							
Brown hair . .		21.976	10.676	14.040	13.319	7.603	6.213
Dark Brown hair . .		22.069	10.703	14.168	13.418	7.523	6.253
Black hair . .		22.138	10.652	14.305	13.333	7.722	6.291

The rule of increased size for dark and decreased for fair followed fairly regularly the shade of the hair, but being liable to minor variations with differences in the colour of the eyes as shown in the foregoing table.

The examples of light-brown-haired Criminals were too few to be put in the table in comparison with the rest, but the averages of those with blue eyes, which may be taken as a specimen, were, for circumference 21.812, and 10.312; for arches 13.937 and 13.312, and for diameters 7.562 and 6.093. For brown and grey eyes the figures were larger.

WEIGHT.

The calculations under this head showed, somewhat contrary to our expectations, that there was a marked and decided enlargement of the head in all directions as the weight of the body increased.

There are, it may be observed, special facilities for inves-

tigating this point in a prison, as probably among no other class of men is the relation between the weight and development of the body so near normal. Not one prisoner in a hundred comes in with a weight above what it should be in proportion to his height and age, while those below the average are quickly raised to their proper standard by their sufficient and regular diet.

The first part of the subjoined table gives the average measurements for each 10 lbs. in weight in prisoners varying from 100 to 170 lbs.; the second gives the amount of increase or decrease in each decade, and the third the *average* increase for each additional 10 lbs. in weight.

I.						
Weight in lbs.	Circumference		Arch		Diameter	
	Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
100 to 110 . .	21·364	10·250	13·760	13·072	7·401	6·131
110 „ 120 . .	21·509	10·348	13·871	13·046	7·455	6·110
120 „ 130 . .	21·774	10·585	13·885	13·114	7·519	6·134
130 „ 140 . .	21·993	10·736	14·088	13·369	7·594	6·217
140 „ 150 . .	22·085	10·751	14·132	13·395	7·625	6·230
150 „ 160 . .	22·347	10·824	14·345	13·425	7·755	6·270
160 „ 170 . .	22·484	10·954	14·335	13·603	7·795	6·362
II.						
110 „ 120 . .	+0·145	+0·098	+0·111	−0·026	+0·054	−0·029
120 „ 130 . .	+0·264	+0·237	+0·014	+0·068	+0·064	+0·024
130 „ 140 . .	+0·219	+0·150	+0·203	+0·255	+0·074	+0·082
140 „ 150 . .	+0·091	+0·015	+0·044	+0·025	+0·031	+0·013
150 „ 160 . .	+0·262	+0·072	+0·213	+0·030	+0·130	+0·040
160 „ 170 . .	+0·136	+0·130	−0·010	+0·177	+0·040	+0·092
III.						
Av. increase .	0·186	0·117	0·117	0·111	0·065	0·042

Applying the Cranial Index to the second table we find that the greatest increase in capacity is in the decade from 120 to 130 lbs., and the least in the decade from 140 to 150 lbs., whilst the third table shows an average increase in cranial value of ·414 of an inch for each additional 10 lbs.

HEIGHT.

As the size of the head varies with a man's weight so does it also with his height—the taller the man the larger his head.

Starting with a man of 5 ft. high having a circumference (head) of $21\frac{1}{2}$ ins., we find an increase of about $\frac{1}{12}$ th of an inch for each additional inch in height, until in a man of 6 ft. the average is exactly $22\frac{1}{2}$ ins.

The other measurements increase in somewhat similar proportion. This rule, however, it will be seen from the table, is more liable to variation than the one enunciated in the preceding section.

Height in inches	Circumference		Arch		Diameter	
	Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
60 . . .	21·500	10·410	13·761	13·124	7·404	6·187
61 . . .	21·772	10·647	13·795	13·116	7·477	6·532
62 . . .	21·846	10·663	13·954	13·228	7·555	6·176
63 . . .	21·948	10·679	14·045	13·278	7·578	6·118
64 . . .	22·014	10·705	14·031	13·433	7·598	6·229
65 . . .	22·016	10·594	14·147	13·267	7·608	6·206
66 . . .	22·215	10·757	14·167	13·386	7·713	6·239
67 . . .	22·200	10·765	14·037	13·420	7·662	6·225
68 . . .	22·225	10·753	14·267	13·406	7·222	6·244
69 . . .	22·375	10·973	14·321	13·687	7·745	6·399
70 . . .	22·425	11·000	14·487	13·650	7·743	6·300
71 . . .	21·875	10·812	13·750	13·312	7·296	6·265
72 . . .	22·500	11·625	14·500	14·000	7·750	6·500

The whole of the cases, Criminals and Lunatics, were considered in two groups, those of 68 inches and upwards, and those under 68 inches in height, with the following result.

Class	Age	Height	Circumference		Arch		Diameter	
			Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
<i>Insane.</i>								
68 inches & over	43·844	68·956	22·346	11·720	14·450	13·627	7·821	6·284
Under 68 inches	45·561	64·261	22·175	11·651	14·125	13·455	7·720	6·246
Difference .	− 1·717	+ 4·695	+ 0·171	+ 0·069	+ 0·325	+ 0·172	+ 0·101	+ 0·038
<i>Criminals.</i>								
68 inches & over	31·430	65·337	22·271	10·855	14·289	13·507	7·704	6·394
Under 68 inches	29·966	63·887	21·984	10·669	14·037	13·306	7·595	6·222
Difference .	+ 1·464	+ 1·450	+ 0·287	+ 0·186	+ 0·252	+ 0·201	+ 0·109	+ 0·172

The above table shows that the differences for those of 68 inches and over, and those under 68 inches, were most marked in the Criminals in all dimensions except the antero-posterior arch.

DISEASE.

For the various diseases of Insanity the dimensions, &c. were as follows :

Disease	Age	Height	Circumference		Arch		Diameter	
			Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
Idiocy . . .	23·210	57·625	21·684	11·342	13·769	13·348	7·434	6·161
Imbecility . .	33·745	65·013	22·054	11·731	14·263	13·522	7·671	6·221
Dementia . .	48·149	65·193	22·144	11·581	14·012	13·353	7·707	6·219
General Paralysis . }	44·290	66·382	22·027	11·441	14·035	13·445	7·761	6·263
Epilepsy . .	37·236	64·361	22·246	11·682	14·334	13·533	7·738	6·257
Chronic Mania	50·72	65·239	22·342	11·738	14·311	13·520	7·780	6·273
Acute forms .	44·371	65·625	22·292	11·736	14·224	13·565	7·784	6·285
Mono-mania of Susp. . }	40·869	66·954	22·260	11·706	14·309	13·543	7·812	6·225

The above figures show that Chronic-Mania had the greatest average circumference both whole and frontal; Epilepsy the greatest antero-posterior arch ; Acute forms the greatest transverse arch and transverse diameter; and Mono-mania of Suspicion the longest antero-posterior diameter.

The Cranial Index arranges the diseases in the following order of skull value :—Chr. Mania, Epilepsy, Mono-mania of

Suspicion, Acute forms, Imbecility, Dementia, General Paralysis, and Idiocy.

IDIOCY.—Several of the members of this class had heads too small to fill the ‘conformateur,’ and consequently we have little new to say about them. It may be noticed, however, that not one of them had a head circumference of less than $20\frac{1}{2}$ inches, whilst one hydrocephalic idiot had a circumference of 25 inches—the largest head in the tables. Only two of the idiotic heads were noticeably asymmetrical, and in one of these cases there was a history of forcible instrumental delivery. This head is shown at fig. 8 in the plate.

IMBECILITY was associated with marked symmetry of skull, the only deviation being a slight tendency to left-headedness. The cases were also remarkable for great width of forehead and anterior half of skull, over 30 per cent. having the greatest transverse diameter anterior to the central point of the head.

The particulars respecting regional development in each class of disease are clearly set forth in the table below; and we only think it necessary to point out the large extent to which the *insane type* of skull prevailed in General Paralysis, 19·4 per cent. of them presenting this peculiarity.

Percentage of									
Disease	Heads larger on			Two sides equal	Greatest transverse diameter in				
	Rt. side	Lft. side	Rt. side in front		Ant. 3rd	Middle 3rd	Post. 3rd	Ant. half ¹	Post. half ¹
Idiocy . .	2	2	2	2	5·3	78·9	15·8	15·8	84·2
Imbecility .	7·1	67·9	7·1	25·0	5·4	92·8	1·8	30·4	69·6
Dementia .	3·0	87·4	17·0	9·6	7·8	77·2	15·0	21·0	79·0
General } Paralysis }	3·0	97·0	16·0	...	19·4	67·7	12·9	35·5	64·5
Epilepsy .	4·0	92·0	18·9	4·0	12·2	78·4	9·4	43·8	56·2
Chr. Mania	9·2	81·6	9·2	9·2	8·2	80·4	11·4	14·4	85·6
Acute forms	12·8	76·8	5·5	10·4	3·7	82·8	13·5	14·7	85·3
Monomania } of Susp. }	17·3	69·7	8·7	13·0	...	100·	100·
Total av. } percentage }	8·057	81·771	11·771	10·171	7·75	82·275	9·975	21·95	78·05

¹ Insane type.

² No percentage taken, as some of heads were too small for ‘conformateur.’

The percentage of skulls of 23 inches and more in circumference in the various diseases was, Idiocy 5·263 per cent. Imbecility 20·° per cent. Dementia 18·660 per cent. General Paralysis 12·5 per cent. Epilepsy 19·230 per cent. Chronic Mania 20·618 per cent. Acute forms 25·443 per cent. Monomania of Suspicion 21·739 per cent. Compare this with percentage of Criminals' skulls of 23 inches and more in circumference, which only reached 6·4 per cent.

NATIONALITY.

Observations on this point were only made upon Prisoners, no exact note being taken of the nationality of Lunatics.

Amongst the men admitted to the Wakefield Prison there are a few Scotch and Welsh, and an occasional foreigner, but the majority are either English or Irish, and in the proportion of about 2 to 1. A table is given below showing the average measurements of all the nationalities that have come under our notice, but only those of the English and Irish must be taken as representing the probable truth; the others are simply given for what they are worth, and must be left for future observation to verify or correct.

Nationality	Circumference		Arch		Diameter	
	Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
English	21·992	10·665	14·094	13·405	7·569	6·230
Irish	22·013	10·621	14·172	13·351	7·611	6·188
Scotch	21·833	10·750	14·166	13·416	7·645	6·083
Welsh	22·458	10·416	14·833	13·708	7·708	6·416
English and Irish ¹ .	22·437	11·000	14·312	13·468	7·750	6·296
Scotch and English ¹ .	22·125	10·500	14·250	13·250	7·750	6·250
Scotch and Irish ¹ .	21·875	10·375	14·250	13·250	7·562	6·125
Italian	22·375	11·000	14·750	13·750	7·687	6·125

Confining our attention to the two nationalities which are first on the list, we find that the Irish have a greater entire circumference and a less frontal; a greater antero-posterior arch and a less transverse, and a similar relationship between the diameters.

¹ Parents of different nationalities.

The Irish head, therefore, has a less frontal development, is shallower and narrower, but longer than the English. The antero-posterior diameter of the Irish, it may be noted, is exactly the same length longer than the English as the transverse diameter is shorter. On applying the Cranial Index to the two classes, however, the Irish preserves its superiority as regards capacity.

This superiority in size of the Irish skull agrees with observations made upon the brain weight of the Insane with reference to their religious persuasion (page 25 of this volume), in which the Roman Catholics, the great majority of whom were Irish, were shown to have the heaviest average brains.

SANE AND INSANE.

All the members of the insane class whose employment had, previous to admission to the Asylum, been of a character requiring a certain amount of head-work and not associated with manual labour—clerks, schoolmasters, master shopkeepers, &c.—were grouped together and gave the following average measurements, which are compared with those of 84 sane individuals of the same or a superior class including doctors, lawyers, clerks, attendants, &c.

Head-workers	Circumference		Arch		Diameter	
	Whole	Frontal	Ant.-post.	Transverse	Ant.-post.	Transverse
Insane . . .	22·461	11·807	14·413	13·660	7·889	6·339
Sane . . .	22·388	11·678	14·313	13·709	7·805	6·316
Difference .	+·073	+·129	+·100	-·049	+·084	+·023

The above table shows the insane to have larger head measurement than the sane in all directions save that of the transverse arch.

NOTES
ON THE
PATHOLOGY OF GENERAL PARALYSIS
OF THE INSANE.

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LORD CHANCELLOR'S VISITOR OF LUNATICS.

NOTWITHSTANDING the large amount of attention bestowed upon it during the last half-century, general paralysis of the insane may still be regarded as an obscure disease, uncertain as to its origin, indeterminate in its nature, and intractable under treatment. Important advances in our knowledge respecting it have undoubtedly been made since it was first clearly differentiated by Bayle, Calmeil, and Delaye, and yet it may be doubted whether in the preliminary process of the diagnosis of this disease, satisfactory precision and uniformity of practice have yet been attained to. Dr. Drouet, of the Parisian Asylum at Ville Everard, thinks that in France it is still sometimes confounded with chronic alcoholism, epilepsy, hysteria, locomotor ataxy, tumour of the brain, encephalitis, and muscular atrophy, and facts are not wanting to suggest that in this country, also, it is occasionally but imperfectly recognised. Such a fact is the appearance of general paralysis in the table of the causes of death in more than one lunatic asylum report, as the sole form of organic disease of the brain having a fatal issue. We know how largely responsible for the mortality of an asylum

general paralysis is, but we know also that there are other forms of cerebral degeneration and disease which invariably swell that mortality, and when we find no recognition of these other forms we are justified in concluding that the term General Paralysis is employed in far too comprehensive a sense.

What is really wanted to a more complete acquaintance with general paralysis than we at present possess, and to some mastery over it, for of that even we need not despair, is an accurate study of its symptoms, a digest of the many valuable but scattered observations on it that have been made, and a series of therapeutical experiments more elaborate and persevering than any that have been hitherto attempted. Graphic sketches of general paralysis abound in medico-psychological literature. Admirable descriptions of its several stages have been given by many authorities, and perhaps there is no form of mental disease that has been more minutely watched, or more vividly depicted. And yet we are without a strictly scientific knowledge of its progress. If we select any one prominent symptom, such as the alteration in the pupils or in speech, and endeavour to ascertain its point of origin, its character, and its significance, we shall find that the widest discrepancies exist in the views of various authorities upon all these points. The procession of the symptoms, their range and combinations, have not yet been traced out with scientific truth, and there do not exist, as far as I am aware, any reports of cases in which daily records have been kept of the successive advances of this incessantly progressive malady, throughout its whole course. But such reports are essential to a genuine understanding of the disease, and until they have been provided we shall have no sure footing for our generalisations regarding it. Until general paralysis has been pursued from first to last, in a great number of instances, with as minute and constant observance as is now bestowed upon acute diseases, until all variations in its mental and motor symptoms have been gauged in many cases with as much care as is bestowed upon the analysis of the urine in diabetes, we shall not attain to a clear insight into its nature nor be in a position to control it

thoroughly. The reports of cases which are at present available are too brief and general. The best of them are wanting in detail; the worst are utterly vague and valueless. In some records of cases it is quite curious to notice with what perverse consistency all that is important is left out of the history of cases of general paralysis, while all that is irrelevant and trivial is inserted. No information is afforded as to the march, of the delusions or dementia, or as to the extent and degree of muscular impairment, but changes in occupation and diet, accidental bruises, or bed-sores, are very circumstantially set forth. Surely this ought to be amended altogether. Scientific requirements, and not statutory provisions, should regulate the preparation of the biographies of diseases, which should be founded on correct personal observation, and not on dubious gossip.

There is one obstacle that must for a time seriously interfere with the acquisition of such full trustworthy histories of cases of general paralysis as we desiderate, and that is the non-recognition of the disease in its earliest and probably most instructive stage. It is generally well advanced before it is identified. It has often existed for many months before asylum treatment is resorted to, and thus but few opportunities are afforded of noting its beginnings and primal growth. But the thorough investigation of it when it does come under observation, which is now advocated, will tend to remove this obstacle to our complete acquaintance with it, for its phenomena are not isolated but are linked together, and when we have once got a firm hold of the chain, we shall be able gradually to trace it back to its starting-point with more and more confidence. It may already be shown, I think, that some of the groups of symptoms have a definite physiological order of development, so that one symptom may be followed up to another symptom out of which it has grown, and thus that every fact in the history of the disease which we establish is a vantage ground from which we may with more exactness proceed to antecedent facts.

But even at present, without waiting for those complete histories of cases which are so much needed, much light might, I think, be thrown upon general paralysis by a diligent

utilisation of the materials that lie to hand. Abridged histories of cases are abundant enough, and bristle with suggestive facts. And then there have been several independent and valuable investigations into particular aspects, features, and relations of the disease. There have been, for instance, Dr. Mickle's investigations into the temperature of the body during its progress and complications, Dr. Clifford Allbutt's and Dr. Aldridge's investigations into the changes in the expansion of the optic nerve which are characteristic of it; Mr. Thompson's investigations into modifications of the pulse form as revealed by the sphygmograph in its several stages; Dr. Fothergill's investigation into alterations in the cardiac sounds during its progress; Dr. Westphal's investigations into its connection with *labes dorsalis*; Dr. Lockhart Clarke, Dr. Gray, Dr. Major, and Dr. Batty Tuke's investigations into its histology; Dr. Merson's investigations into its urinology; Dr. Burman's into its statistics, and Dr. Newcombe's into the conditions of its epileptiform seizures. These investigations, and others that might be mentioned, have provided a store of facts that may be so dealt with as to yield inferences of great value. After eliminating from the results of the labours of each investigator whatever is hypothetical or in dispute, there will remain a residuum of established fact, that will justify us, if not in constructing a theory of the disease, at least in discarding peremptorily a number of crude notions respecting it that have been inconsiderately advanced, and that have helped to prolong the ignorance that led to their toleration. To get rid of error is the first step towards the attainment of truth; and if a collocation and critical analysis of the researches that have been prosecuted into the natural history of general paralysis had no higher result than to clear the way of obstructive speculations, they would well repay the trouble expended on them. It is to be anticipated, however, that they would do more than this, that they would afford guidance for future explorations, and would at once add much to our positive knowledge of the disease.

And our positive knowledge of the disease might also be advanced to no inconsiderable extent, by the therapeutic

experiments that are so urgently demanded in order that we may more effectually control its progress. I think it will be admitted that general paralysis is now very commonly regarded as an altogether irremediable malady, and that a majority of those suffering from it are not submitted to any kind of medical treatment. And yet no very conclusive reasons can be adduced for the despairing view that is taken of it. Quite recently, Dr. Broadbent has published a series of cases illustrative of syphilitic affections of the brain, in two of which most of the symptoms of general paralysis, mental and bodily, were present in a very pronounced manner, and in which complete recovery took place under treatment by iodide of potassium and the biniodide of mercury. I am satisfied that these two cases, if they had found their way to an asylum, instead of to Dr. Broadbent's consulting room, would have been classified as cases of general paralysis, and indeed a very fair show of argument might be made that they were really of that nature. Well, in these two cases recovery took place, a fact which sufficiently indicates the grave impropriety of foregoing treatment whenever the symptoms of general paralysis are present. For it was the treatment in these cases that secured the recovery. When it was interrupted, relapses took place; when it was steadily persevered in, improvement went on. Had no treatment been attempted, death, and not recovery, must have been their termination. Are we, then, justified in leaving without treatment another group of cases presenting the same symptoms because we have given them a different name, and have formed, perhaps, a different theory as to their essential nature? At no very remote period, Dr. Broadbent's cases would have been looked upon as hopeless, and would have been left to sink deeper and deeper into the slough of despond under the belief that they were instances of softening of the brain. Is it not possible that our professional descendants may look back with pity and censure upon the helpless attitude that we have been content to assume in the presence of general paralysis? Whilst we are folding our hands and waiting for fuller information about that disease, it is destroying its hecatombs of victims,

and other diseases not unlike it have been restrained in their ravages. And the disease itself mocks our inactivity, for occasionally it undergoes something very like spontaneous cure. The symptoms abate and are permanently arrested. More frequently the onward march of the disease is temporarily stayed. But such arrests, temporary and permanent, hold out a prospect of modifying the course of the malady by means of treatment, and mitigation and protraction of a morbid process is the next best thing to its abolition.

The revolt against a blind empiricism in the treatment of disease, which was laudable in itself and has had many happy consequences, has perhaps carried us too far into the regions of blank expectancy. We are too apt to demand a physiological passport of every proposed remedy, and to discountenance the treatment of symptoms. But surely, in utter darkness, it is better to grope about lest, perchance, a lamp might be found, than to wait in inactivity in the hope of a light being brought. Surely in the case of a very fatal disease it is better to treat symptoms than to treat nothing. For to subdue a symptom is sometimes to break up a morbid confederacy, and to modify a symptom is sometimes to obtain a clue to the seat and nature of morbid action. With our improved knowledge of drugs they are becoming prisms by means of which we may analyse the manifestations of disease, and acquaint ourselves with the composition of remote morbid processes. And hence the employment of drugs in general paralysis may enable us to influence its course beneficially and may give us an insight into its nature. The former power over general paralysis possessed by drugs is illustrated by the action of the iodide of potassium and biniodide of mercury in retarding its advance. The latter may be illustrated by the action of quinine, which has been shown by Dr. Hammond to induce cerebral hyperæmia, and which, when given in general paralysis in large doses, brings on paroxysms of violent excitement.

Valuable results may, I think, be anticipated from a patient and extensive trial of Calabar bean, of arsenic, of veratria viride, of chloral combined with digitalis, of belladonna, of muscarin and other drugs in general paralysis.

My present object is not, however, to describe any such trials, nor even to indicate the direction which they might most hopefully take, but to record some observations on the disease, to indicate the conclusions which these observations appear to warrant, and the further enquiries which they suggest.

The brain of a general paralytic patient who has died in the ordinary course of the disease, at the end of the third stage, generally, with its envelopes, presents, to a superficial examination, certain appearances that are distinctive. Those who have had much experience in the *post-mortem* theatre of a lunatic asylum ought to be able to pick out the fresh brain of a general paralytic from amongst a number of other brains of chronic lunatics. Occasionally in making such a selection they might be at fault, for in some brains of general paralytics the distinctive appearances are not well marked, and in some other forms of brain disease, such as chronic atrophy and chronic alcoholism, the appearances presented very closely resemble those seen in general paralysis. As a rule, however, the brain of the general paralytic should be fixed upon without much difficulty, but the characteristic and distinctive appearances which enable it to be thus recognised are more easily seen than described. Indeed, when they are enumerated one by one they cease to be distinctive, for each of them is encountered in other forms of brain disease having no alliance with general paralysis. There is not one of them that is peculiar to that disease, and it is in the grouping and combination that the distinctiveness consists.

Let us suppose that the brain of a patient who has died of general paralysis has been removed from the skull, and that in this case there are no arachnoid cysts nor other adventitious conditions. What first strikes us is an abnormality in the shape and consistence of the brain. It is somewhat attenuated in the frontal region, and it lies upon the board as if it were flattened and collapsed. It lacks the rounded contour and plumpness of the healthy brain, and has an uneven outline and lateral bulgings, obviously due to the softness of its texture, which renders it unable to

preserve its proper form when deprived of the support of the cranial walls. All fresh brains of course, except those that are sclerosed, fall away to some extent when they are laid upon a flat surface, but the subsidence of the general paralytic brain is greatly in excess of what is ordinarily seen. And not only is the brain softened, but it is atrophied, and there is an obvious decrease in the volume of the entire cerebral mass, as may be ascertained by exact measurement and comparison with a healthy standard, and as might have been inferred from the large amount of serous fluid with which the brain is bathed. In no form of mental disease, except in the dementia of brain wasting, is atrophy of the brain so advanced as in general paralysis, which is all the more remarkable, as death happens in general paralysis in that decade of life in which the maximum weight of the brain is attained.

The next observation that occurs to us is that the arachnoid is greatly thickened, and that it has in many places lost its transparency, and become white and opaque. Along the margins of the great median cleft, where the Pacchionian granulations are unusually numerous and of large size, it has become a tough, glistening membrane, and over the frontal and parietal lobes it is in many places cloudy or opalescent. Over the occipital lobes the arachnoid retains its natural aspect, but over the temporo-sphenoidal lobes and cerebellum, scattered patches of whitish opacity are generally to be noticed. These are, however, small and few in number when compared with the patches of thickening and opacity over the upper and frontal aspects of the cerebrum. Wherever situated, the opaque patches are best seen over the sulci and not on the summits of the gyri. In addition to these patches lines or belts of similar whitish opacity may sometimes be noticed running parallel to the vessels on each side of them, conforming to all their sinuosities. Directing our attention next to the pia mater, we notice that it as well as the arachnoid is remarkably thickened. Instead of being thin and delicate, it is coarse and tenacious, and has a swollen and somewhat gelatinous appearance, owing to infiltration of its meshes with serous fluid. It is not, however, pale and

watery, as in brain-wasting, but is irregularly hyperæmic, presenting amidst its oedematous expanse reticulations of dilated vessels, and with more or less injection. The distribution of the morbid changes in the pia mater corresponds with that of those in the arachnoid. They do not extend on to the occipital lobe, and are most marked over the frontal and parietal lobes. As regards the great arteries of the brain, we are struck by their freedom from atheromatous deposit and calcareous degeneration. A few specks or blotches of atheroma may be seen on the basilar or anterior cerebral arteries, if the patient has been attacked late in life, but these are not at all of the character of the changes noticed in the arteries in senile or organic dementia. Looking next at the convolutions as seen without disturbing the membranes, a glance suffices to show that they are much atrophied, the wasting being most advanced in the frontal and parietal lobes, present to some extent in the upper part of the temporo-sphenoidal lobes, but invisible in the occipital lobes or orbital lobules. To compensate for the wasting there is much oedematous effusion, which is diffused throughout the meshes of the pia mater, and collected in some places in hollows and channels, where the wasting is very far advanced. The wasting of general paralysis seems to differ from that of senile, simple and consecutive dementia, in that it is nearly as well marked in the parietal as in the frontal lobes, and to agree with it in that it does not invade the occipital lobes. From the wasting of organic dementia it may be readily distinguished because it is less localised, and because there is no rusty brown staining of the membranes at the bottom of the deepest depression, due to destruction of cerebral substance.

The appearances now enumerated as visible on the mere outward inspection of the brain of a general paralytic patient, are of themselves often sufficient to the practised eye to reveal the nature of the disease, and I have repeatedly seen my colleagues at the West Riding Asylum determine with accuracy, by the presence or absence of the mere outward appearances, whether or not a brain submitted to them, of the history of which they knew nothing, had belonged to a patient

affected by general paralysis. But there are other appearances in the outer coverings and in the texture of the brain that are very characteristic of the disease. The skull is generally thickened, but not of very dense consistence, its inner surface having a reddish-blue tinge, owing to the presence of blood in the diploë. Sometimes the skull is decidedly soft, and in these cases the thickening is considerable, while the grooves for the meningeal vessels are very deep, and have an excavated appearance as if the artery was lodged in the bone, as it actually is, for osseous ledges may sometimes be seen projecting over it and occasionally uniting to form a complete bridge. The dura mater, which is thickened, is adherent to the skull, most so in the frontal region, but here the adhesions are not of that strong and intimate character so often noticed in senile dementia, where the greatest force is required to tear through them, and where shreds of the dura mater—white, glistening, fibrous shreds, often of large size, are left attached to the bone. On the inner surface of the dura mater, which is rather dirty in colour throughout, and the sinuses of which contain dark fluid blood and clots, there is more or less staining of a rusty brownish-yellow colour, which often forms a distinct film and can always be scraped off. This staining, which is most distinct and extensive when there is an arachnoid cyst, may occur in any region, but its favourite habitat is the temporo-sphenoidal fossa. Besides this rusty staining there are seen on the inner aspect of the dura mater the remains of filiform bands which have at certain points passed between it and the pia mater.

If the dissection of the brain be next proceeded with, the thickening of the pia mater is again impressed upon the observer when it has to be torn through, and a new pathological feature comes into view, when the attempt is made to strip it off. It is then found to be in some places intimately united with the grey matter of the convolutions. Instead of coming away from the frontal and parietal lobes, as a loose and well-worn glove does from the hand, as in most cases in which there is wasting of the brain it does, it is glued to the surface of certain gyri, like a glove that has dried into

an open sore, and if it is forcibly torn away it removes some tissue, and leaves a rough eroded surface beneath. The superficial layers of the cortical substance are, in fact, torn away with the pia mater, and may be seen adhering to its inner aspect. The points or areas of adhesion may be few or numerous, large or small, distinct or confluent, but they are invariably confined to the summits of the gyri, and do not spread down their sides nor occur at the bottom of the sulci. They have an irregular outline and jagged edges, and incline perhaps to an oval shape, having their long diameter parallel to that of the convolution on which they are situated. When the pia mater has been stripped from a region of the cerebrum in which the adhesions are abundant, the denuded and abraded convolutions look just as if they had been gnawed and eaten away by a caterpillar. The surface of the convolutions, where not implicated in the adhesions, is generally smooth and normal. Occasionally, however, in a few places, it has a granular, or rather, perhaps, a porous appearance, as if it were perforated by numerous minute holes. It is always paler than the eroded patches, which are darker than the cortical substance usually is, and have often a red and engorged aspect. The removal of the pia mater makes more obvious the wasting of the brain already adverted to. The gyri are seen to be attenuated, and the sulci greatly enlarged.

On slicing the brain, a general deterioration in its quality is at once recognised. The grey matter has a shallow and faded appearance, and is traversed by vessels that are coarse and prominent, and in some places it has a violet or vinous tinge. It is undoubtedly softened and watery, as is also the medullary substance, which is of a dirty, greyish-white colour, and presents an excessive number of coarse vascular points, and a varying number of blotches of vinous staining. The ventricles, which are abnormally capacious, contain an excessive amount of serous fluid, the choroid plexuses are œdematous, and the ependyma is often granular. The basal-ganglia are all more or less atrophied, and the cerebral nerves as a rule have a preternaturally white and glistening appearance.

In the case of a brain in which all the pathological

appearances now enumerated were markedly present, there would be probably no great difference of opinion as to the nature of the disease causing death. I do not know of any disease save general paralysis in which all these appearances, and no others, might be found. But the difficulty is that in a large number of cases of undoubted general paralysis these appearances are not all found, and that, in a small number of cases, some other appearances, which have not been here described, are present. But this difficulty is not peculiar to the study of general paralysis, for in almost every fatal disease there are extensive variations in its morbid anatomy. The question that arises in view of this difficulty is, can we fix upon any one appearance, or group of appearances, as being invariably present? and in reference to general paralysis the answer to that question has been hitherto given in the negative. All authorities are agreed that there is no appearance that is constant, and that can therefore surely guide us to the pathological starting-point and nature of the malady, and most authorities are agreed that there is no appearance nor group of appearances that is so pre-eminently frequent as to be entitled to paramount attention, or to be regarded as the central fact in a circle of pathological changes. In differing from most authorities on this latter point, and in maintaining that there is one morbid appearance in general paralysis that is so frequent as to be specific, I rely upon a very large number of observations made during the last ten years, and upon the facts revealed by certain methods of examining the brain that have not, I believe, been adopted by other investigators.

The one appearance in general paralysis, it seems to me, which is very constant and very characteristic, is the adhesion of the pia mater to the cortical substance. This appearance, which was originally discovered by Bayle,¹ and was regarded by him as corroborative of his theory of the disease, which he worked out with much ingenuity, viz. that it is analogous to chronic meningitis, has been since noted by everyone who has investigated the anatomical conditions of

¹ 'Recherches sur l'abracnitis Chronique,' Paris, 1822, p. 25, quoted by Foville.

general paralysis, but has mostly been set aside as an accessory or inessential feature. Its real significance has been overlooked, I think, because all hopes of penetrating the mystery of general paralysis have for many years past been fixed on the microscope, and because in the scant attention bestowed on the hand-and-eye examination of the brain, its presence has often escaped detection. So long as I continued to examine the brain in the usual way, and to test the relations of the pia mater to the subjacent grey matter, only at a few points selected at haphazard, so long did I share the prevailing belief that the adhesions between these were only occasional and secondary. It was only when, in pursuance of some anatomical researches, I carefully stripped the whole brain in many cases of general paralysis, that I came to recognise the frequency of adhesions more or less extensive in some region of the cerebrum. Since the frequency of these adhesions was first recognised, I have given them a large amount of attention, and have come to these conclusions: 1st, that they constitute the most constant pathological change in general paralysis; 2nd, that they explain the essential nature of the morbid process in that disease; 3rd, that they will also, when minutely studied, explain its symptoms and progress.

That they are the most constant change will scarcely be disputed, but that they can only be said to be constant and not invariable, may be regarded as fatal to the expectation that they will explain the disease. If cases of general paralysis occur—cases presenting all its familiar symptoms and running its accustomed course, in which, after death, no adhesions are discoverable, and if cases of other forms of cerebral derangement occur—cases presenting symptoms different from those of general paralysis, in which, after death, adhesions are found, then in what sense, it may be asked, can adhesions be said to have any necessary connection with the morbid process peculiar to the disease, whatever that may be? The question is a very pertinent one, and unless it can be satisfactorily answered, it would be useless to concentrate attention upon these adhesions, and to endeavour to connect them with special symptoms. In endeavouring

to answer it, I would in the first place point out that the proportion of cases of general paralysis in which adhesions when carefully looked for are not present, although still not definitely ascertained, must be exceedingly small. Judging from my own more recent experience, I should say that adhesions exist in 80 per cent. of all cases of general paralysis. At one time I should not have alleged that they occurred in so considerable a proportion, but the more thorough inspection of the brain which, in such cases I have lately instituted, has led me to see that in some instances in which I should formerly have ignored them they were present very unmistakably. And it is only after a thorough, laborious and time-consuming examination of the brain that we are entitled to say that it is free from adhesions, for, as I shall presently show, they are sometimes very irregularly distributed and narrowly localised. It is not enough to have stripped a lobe or a hemisphere. Unless the whole brain has been denuded we are not justified in saying that there are no adhesions. Now, there can be no impropriety in asserting that, in many cases in which adhesions have been declared absent, this complete denudation, which besides being tedious, demands some manipulative skill, has not been carried out. An exploration has been made of the region in which adhesions usually occur, and because they have not been found there it has been inferred that they do not exist elsewhere. But this inference is quite unwarrantable. Some twelve months ago I examined a brain in which, by experimental stripping with the forceps under water, I could not discover any adhesions, which were, however, revealed in considerable numbers when the brain was examined by another method which I shall presently describe. When that method is generally adopted in the examination of the brains of general paralytics, it will, I think, be admitted, that adhesions between the pia mater and cineritious substance exist in 80 per cent. of all fatal cases.

In twenty out of every hundred cases of general paralysis, however, it is admitted that adhesions are not recognisable, that is to say if these twenty cases have been correctly diagnosed as instances of general paralysis—a qualification which

is very needful—because, as I have already pointed out, the diagnosis of the disease is not always accurate. And curiously enough, in my own experience, it has just been in those cases in which diagnostic doubts have intruded during life that the adhesions have been wanting after death. The real difficulty seems to me to be to distinguish general paralysis from chronic atrophy of the brain; and it has invariably been in those cases of general paralysis that have most resembled chronic atrophy of the brain, that the absence of adhesions has been noted. It is a fact, and a very significant one, that the adhesions are always most plentiful and firmest in the most typical and acute cases of the disease. If the delirious conceptions have been of the most extravagant kind, if the motor disturbance has been severe, if the excitement has been intense and the febrile action considerable, if the course of the disease has been rapid, then adhesions will be abundant. But if, on the other hand, the chief manifestation of the disease has been a gradual, slow diminution of the intellectual powers, with ill-defined motor impairment, and without very prominent delusions or great emotional perturbation, then the adhesions will be few in number or may be altogether absent. That there are varieties of general paralysis is universally acknowledged, and that it is in what may be called its mildest variety, viz. that bordering most closely upon simple brain-wasting, that the adhesions of the pia mater to the grey matter are most frequently absent, is certainly the result of my experience.

The circumstance that it is in protracted cases of the disease that the adhesions are most frequently absent, is worthy of consideration in another connection; for it is possible that in such long drawn-out cases, the adhesions may have existed at one time and may have been broken down. If we suppose that these adhesions result from a continuity of inflammation or infiltration of the pia mater and the surface which it clothes, we can understand how, when inflammatory action has subsided, contraction and absorption may take place, and how, as atrophy of the brain advances, the œdematous condition of the surface which inevitably ensues, may tend to the liquefaction and severance

of adhesions that are not particularly dense. The slightly-roughened, granular, or porous-looking patches that are sometimes seen in close contiguity to existing adhesions in general paralysis, look as if they, too, might have been the seat of similar morbid changes, which had in some way undergone resolution. Magnan¹ found that by injecting water into the carotid artery and internal jugular vein he was able to render existing adhesions easily separable; and although my own experience does not accord with his, for I have not succeeded, by passing currents of hot or cold water, or even of steam, through the vessels in cases in which the adhesions were strong, in removing or perceptibly weakening them without actually breaking down the cerebral substance, it is easily conceivable that prolonged œdema of the pia mater might wipe out these adhesions, especially when they were not originally very strong.

But even abandoning this supposition, and allowing that there are undoubted cases of general paralysis in which there are no adhesions nor any vestiges of them, does it therefore necessarily follow that these adhesions do not represent the fundamental morbid process? Assuredly not. The process of which these adhesions are the result in so large a majority of cases, may, in a few cases, fall short of their production, and their absence in these few cases is not more perplexing than the similar absence of characteristic lesions, often encountered in other diseases. In the fulminant form of cerebro-spinal meningitis, the characteristic anatomical lesions are often absent, and the brain and its membranes look healthy, although they have been the seat of changes which have been quickly fatal, and which cause, in most instances of the disease, structural alterations as pronounced as intense hyperæmia, effusion of serum, gelatinous deposit, and purulent infiltration. Practically, the apparent absence of the ordinary encephalic changes in a few cases of general paralysis may be a fact analogous to that sometimes noted in cases of variolous and scarlatinous poisoning, in which the characteristic eruption of the disease has not been developed.

¹ 'De la Lésion Anatomique de la Paralyse Générale.' Hlèse, Paris, 1866, p. 30.

There are, however, as has been said, other cerebral derangements besides general paralysis in which adhesions of the pia mater to the cortical substance are found, and that fact is opposed to the theory that they are in any way characteristic of the disease. But if we enquire into the nature of those other cerebral derangements in which adhesions occur, we shall find that they are all in some degree allied to general paralysis, and that in each of them there is something distinctive about the adhesions. Having examined upwards of 1,500 morbid brains, I do not remember to have encountered adhesions, excluding general paralysis, except in the following diseases: tubercular meningitis, encephalitis, meningitis, acute and chronic, atrophy of the brain, chronic alcoholism, and syphilitic cerebral degeneration. I am aware that it has been stated that such adhesions have been found in healthy brains, but it seems to me that those who could confuse the difficulty of removing the exceedingly delicate pia mater of a healthy brain with the adhesions in general paralysis must be very ignorant of cerebral pathology. It is not necessary to refer to the adhesions which are often seen in the neighbourhood of tumours, and other localised lesions which are clearly due to pressure and circumscribed inflammatory changes, and which are in no danger of being mistaken for anything else.

Of the cerebral derangements enumerated in which adhesions similar to those of general paralysis occur, I need not say more respecting cerebral atrophy, than that such adhesions do occur in a small number of cases that have been so denominated, and that I am not at present prepared to account for their occurrence under such circumstances, except by what may be regarded as the very gratuitous assumption, that these cases were really instances of general paralysis of the slowly progressive type. In relation to the other cerebral derangements in which adhesions of the pia mater have been found, three important considerations have to be borne in mind. In all of them there is a combination of mental and motor symptoms, to some extent resembling general paralysis; in all of them the morbid process from which the adhesions resulted was of an inflammatory kind;

and in all of them there is something connected with the adhesions distinguishing them from those of general paralysis. In tubercular meningitis, we have confusion of thought, double vision, delirium, coma, inequality of the pupils, ptosis, tremors, twitchings, epileptiform convulsions, and paralysis occurring early in life, symptoms which undoubtedly proceed from irritation or diffuse inflammation of the cerebral substance, and leave numerous adhesions of the pia mater, which are, however, most numerous in the basilar region, depend upon an eruption of tubercles in the vessels, and are associated with yellow exudations and whitish granulations. In encephalitis and meningitis, acute and chronic, we have febrile disturbance, headache, restlessness, excitement, sleeplessness, hyper-sensitiveness, hallucinations, stupor, insensibility, contraction and dilatation of the pupils, spasms, convulsions, paralysis, conditions due to inflammation which commences in the membranes of the brain and spreads to the cortex, or *vice versâ*, and which leaves behind it not only adhesions, but punctiform extravasations, fibrinous exudations, and purulent infiltration, especially along the vessels. In chronic alcoholism and syphilitic cerebral disease, we have symptoms, mental and bodily, which, in the absence of the history of the case, it would often be impossible to distinguish from those of general paralysis, with indications of slight encephalic inflammatory action, and with *post-mortem* changes in the minute vessels which, as revealed by the microscope, are characteristic.

It is particularly instructive to notice that in chronic meningitis, the symptoms of which are sometimes almost identical with those of general paralysis, we have adhesions which would be indistinguishable from those of general paralysis were it not that pus, which is never seen in general paralysis, is here seen in other regions of the encephalon. Is it not thus suggested that in general paralysis there is an inflammatory irritation of lower intensity than the suppurative, and ending in non-purulent products? Does it not seem probable that these adhesions, which, it is argued, are the most characteristic feature in its morbid anatomy, and which clearly depend, when present in other diseases, upon

inflammatory action, here also depend upon an inflammatory process? May not differences in the character of that process, in its mode of origin and accompaniments, account for the differences seen in those adhesions as they occur in general paralysis and in other diseases?

The two most remarkable differences between the adhesions in general paralysis and in chronic meningitis are that in the latter there are deposits of pus in their vicinity, which is not the case in general paralysis, and that the adhesions are widely diffused, while in general paralysis they are more isolated and scattered. The first difference betokens that the inflammatory process in general paralysis is less severe than in chronic meningitis, and that in fact it corresponds with a non-purulent catarrh of an epithelial surface; the second difference indicates that the inflammatory action starts in the cerebral matter and not in the membranes. The tendency of all congestions and inflammations beginning in the pia mater is to become widely diffused, while the tendency of those originating in the nervous substance is to confine themselves to small areas. In the pia mater there is an enormous number of large vessels ramifying through a loose parenchyma which offers the most favourable conditions for the propagation of inflammation and the accumulation of its products. In the cortex of the brain, on the other hand, into which only the finer vessels and those not subject to much variation of calibre enter, the territories of nutrition are very small, and hyperæmias and inflammations are much circumscribed, and are extended by functional irritation rather than by vascular distribution or contiguity of tissue. The scattered patches and numerous isolated areas of adhesions, so often seen in general paralysis, are therefore, in all probability, due to inflammatory changes commencing in the cerebral substance.

And this view of the origin of what I regard as the characteristic pathological change in general paralysis appears to harmonise with what we know of the etiology of the disease. True, the most conflicting notions as to its etiology have been advanced and defended; but if we take such of these notions as are worthy of serious attention and view

them together, we shall find that they may be generalised as abusive functional activity of the brain. There may, perhaps, be a diathetic basis to the disease—upon that we shall not now enlarge—but it seems that there has always been some unwise expenditure of brain power to set the morbid process going. That expenditure may have been through the immoderate exertion of the muscular apparatus, through the indulgence of the sexual passion, or of anger, through inordinate ambition, through unfettered imagination, through uncontrolled grief, through intellectual overwork sustained by stimulants or narcotics; but through whatever channel it has occurred, the waste has always been there. There has been excessive functional activity of some district of the cerebral cortex, with excessive hyperæmia of that district, a hyperæmia identical with the active congestion of glands and membranes when their functional activity is exalted. The hyperæmia is excessive in two ways—it is carried too far and it is too often repeated. The capillaries, which are in immediate contact with the active elements of the brain, are over-distended and lose their elasticity and they are too often called upon for large supplies, and are so deprived of restorative rest. There is a state of over-excitement in certain cerebral centres, and this over-excitement induces hyperæmia, dilatation of arteries, an increased flow of blood through the part, a rise of temperature, and an increased transudation through the capillaries, which again leads on to stasis and then to atony of the vessels. The engorgement of the cerebral vessels, the walls of which are so peculiarly delicate, leads also to punctiform hæmorrhages, which, perhaps, mostly take the form of minute dissecting aneurisms of the smallest veins, and is associated in time with changes in the walls of these vessels themselves, with an exudation of lymph, and with perivascular growth of connective tissue which ultimately spreads through the nervous elements. Parallel with the changes enumerated, there must of course be various disorders of the cerebral functions, passing ultimately into their more or less complete abolition.

This rough sketch of the progress of general paralysis,

which is suggested by its etiology and by analogies drawn from other cerebral diseases the course of which is well known, may be justified as to the accuracy of its outline, and filled in with numerous details by a more minute examination of the disease. The primary assumption that general paralysis is an inflammatory disease, which I have founded upon the similarity of the most striking lesion that it leaves behind, in kind if not in degree, to a constant lesion in meningitis, an unquestionably inflammatory disease, and which I have confirmed by an appeal to its etiology and the probable consequences of its causal conditions, will not, I am aware, pass unchallenged. Bayle, by whom the inflammatory nature of general paralysis was first taught, has been acutely criticised by Westphal, who is altogether opposed to this view of the disease, and whose opinion is entitled to the greatest weight. After fully considering Westphal's arguments, however, I am unable to agree with him, either in his strictures on Bayle or in his own hypothesis of the nature of general paralysis. The inflammatory theory seems to me to be more reasonable and more in accordance with the facts before us than any other theory that has been propounded. That theory has been powerfully supported by Meyer,¹ who has recently endeavoured to establish a connection between the variations of the bodily temperature in general paralysis and in some other chronic febrile diseases. From his observations he concludes that we have to do in general paralysis with an inflammatory process affecting the meninges. Exacerbations of this process are to be recognised by increased maniacal excitement accompanied by elevation of temperature. It has been objected to Meyer's conclusions, however, that he misinterpreted the elevation of temperature in the cases which he observed. It has been alleged that the elevation was probably due to violent muscular exertion, to erysipelas, phthisis, and other intercurrent diseases, or to bed-sores or accidental injuries. Even admitting that this was so, the fact still remains that there are many other

¹ 'Die allgem. Progress. Gehirnlamung, eine chron. Meningitis,' Berlin, 1838. Quoted by Westphal, 'Journal of Mental Science,' vol. xiv. p. 179, July 1868.

observations on record in which, when such sources of fallacy were carefully avoided, a distinct elevation of temperature was still found in general paralysis—just such an elevation as we should expect in this disease, not very great, but sufficient to mark febrile disturbance. The inflammatory action, if it be inflammatory, is slow and insidious in its progress and chronic in character. It takes months, or even years, to spread over the cerebrum, and involves, probably, only one part of the cerebrum at a time. Undoubted chronic inflammations in other organs might be pointed to, in which there is no greater nor more persistent raising of the bodily temperature than in general paralysis. It might be, too, that the irritated condition of the cortex tended to keep down the temperature which the inflammatory process would raise, for Bochefontaine asserts that there are three points at least in the cortex, electrification of which exerts a distinct action on the circulation. The frequency of the pulse is sometimes augmented and sometimes diminished when they are stimulated, and as a rule there is augmentation of the arterial tension, though sometimes the reverse is observed.

Looking at the tracts of adhesion on the brain of a general paralytic, and remembering that at least months have been expended in the production of them, we should indeed be surprised to be told that any such rise of temperature had been induced by the low and slow inflammatory process that induced them, as is demanded by the opponents of the inflammatory theory, as a condition of its acceptance. There is in general paralysis, as before stated, just that rise of temperature which we should expect in a chronic inflammatory process in the cortex, and that rise in temperature occurs just at those stages and epochs in the disease when the inflammatory action may be supposed to be most active. It is most constant and decided in the earlier stage, and unless there are intercurrent diseases it does not occur in the last stage, when inflammation has given place to atrophy. Dr. Mickle has shown that a rise in temperature often accompanies a maniacal paroxysm, and precedes convulsive and congestive seizures. He has also shown that a rise in temperature may

occur during the course of general paralysis without any apparent alteration in the mental or physical state.¹

An interesting observation of Dr. Merson's,² that there is an increased elimination of urea in general paralysis, points, I think, in the same direction as the elevated temperature,—towards an inflammatory process in the cortex. This excess of urea excreted betokens an increased disintegration of the protein compounds of the organised tissues, which is just what Moleschott has proved to result from functional hyperæmia and irritation of the brain.

There is a symptom also frequently present that strongly suggests inflammatory action involving the membranes, and that is headache, which is sometimes so severe as to induce the patient to beat his head, to knock it against the wall, or to rub the scalp until it is denuded of hair. This headache, which occurs mostly in the second stage of the disease, is seated in the frontal or parietal region, and is accompanied by giddiness and a sense of fulness and pressure within the skull. Now, without committing myself to the view taken by some very eminent authorities, that the cerebral substance is, under all circumstances, absolutely insensitive, I think I may safely say that headaches of the kind just mentioned almost certainly depend upon irritation of the membranes, which are very sensitive indeed.

But there are anatomical facts, as well as symptoms, which support the opinion that general paralysis is an insidious inflammatory disease. The brains of patients dying early in the disease have been occasionally examined, and then the whole brain has been seen to be congested, while the cortical substance, in certain districts, as well as the pia mater, was swollen and engorged. Throughout the brain, or in some districts of it, minute red spots have been seen in greater or less numbers, and incipient adhesions of the pia mater have also been observed. These adhesions have not been of the dense character with which we are so familiar. The membrane has stuck to the grey matter in certain territories and has been

¹ 'The Temperature in General Paralysis of the Insane,' by W. Julius Mickle, M.D. 'Journal of Medical Science,' vol. xviii. p. 31, April 1872.

² 'The Urinology of General Paralysis,' by John Merson, M.A., M.D., 'West Riding Lunatic Asylum Medical Reports,' vol. iv. p. 63. London, 1874.

difficult to separate, but with care and the gradual rupture of connecting bands and filaments, it has been removed without lacerating the cerebral cortex. The grey matter in these cases is of a reddish colour, and, together with the medullary substance, looks as if it had been sprinkled with wine, which has caused blotches of discolouration. The vinous tinge is most visible in the middle layer of the cineritious substance. Occasionally, also, a viscid gummy substance has in this stage been detected, smearing or permeating the cortical substance. And these appearances, although not very striking, are all that might be anticipated in a brain that had been partially affected by an inflammatory process of the subdued and non-purulent type that has been indicated. We might, indeed, have encephalic inflammation without even these equivocal records. Dr. Wilks has said that cases are sometimes encountered which suggest urticaria of the brain, although this may sound a little like nonsense at first. Of course, if there were such a condition as urticaria of the brain, we should not expect to see any traces of it after death, any more than we expect to find ocular proof of urticaria of the skin after death.

The appearances noted when death occurs in the latest stage of general paralysis which have been already described, are even more corroborative of its inflammatory character than those seen when it is cut short in its career. The thickening of the skull is a consequence of often-recurring congestion of the head; the thickening of the dura mater, its discolouration, and the false membranes which line it, are the vestiges of a past pachimeningitis. The bands passing between the dura mater and arachnoid are shreds of lymph or blood-vessels proceeding to a false membrane in which they break up into stellate ramifications. The thickening and opacity of the arachnoid, the thickening and œdema of the pia mater, and particularly the double streaks of whitish yellow thickening following the vessels, are very characteristic of a past lepto-meningitis. And the general atrophy of the brain, the pappy and softened condition of its substance, the pallor of the cortex, the dilated and tortuous vessels with thickened walls, and the copious effusion of

serum, are the very changes which we should look for in a brain that has been dilapidated by slow inflammatory encroachments.

Then the microscopic appearances, when interpreted, have the same meaning as the cruder morbid anatomy. These are, as regards the membranes, distinct lamination of the two widely separated layers of the pia mater, distension of the minute vessels, in some instances hypertrophy of their muscular coat and the presence of a finely hyaline substance around them, with deposits of hæmatoidin, or the *débris* of extravasations; and as regards the nerve substance, dilatation and tortuosity of vessels, and great distinctness of the hyaline sheaths, abundant deposits of hæmatoidin and pigment, inflation, or shrinking, and disorganisation of the cells, with accumulations of molecular *débris* round their nuclei, and particles of molecular degeneration in the white substance. And these appearances are trustworthy records of former hyperæmia, long continued, often repeated, merging into inflammation of a low non-purulent type and ending in degeneration. From the hyperæmia there will, in all likelihood, ultimately result a gelatinous exudation on the surface of the cerebrum, similar to what is seen in acute hydrocephalus and in meningitis. This exudation we may suppose glues together the pia mater and the tops of the gyri, and in it certain changes are gradually wrought, such as vascularisation and transformation of embryonic into fibrous connective tissue. At the same time in the brain substance, the hyperæmia has induced another train of events, probably increase of perivascular protoplasm, thickening of the vascular walls, nuclear proliferation, and the development of an anastomotic network of connective tissue corpuscles, causing impaired nutrition and more or less disintegration of the nerve elements.

But in dwelling upon the importance of hyperæmia in the origination of that series of changes in which general paralysis consists, and of vascular changes in the furtherance of its disastrous career, it is not to be lost sight of that its true fountain is in the nervous system.

Meschede has done good service in recalling attention to the ganglion cells as the real source of the malady, and

whether or not he prove correct in thinking that he has made out a parenchymatous inflammation of these, there can be no question that he is right in arguing that changes in these cells correspond with the various stages and exacerbations of the disease, and that it is their gradual destruction that involves the gradual abolition of mental life. The greater facility with which morbid modifications of the cerebral membranes, blood vessels, and connective tissue can be made out has conduced to an undue magnification of the share they take in the production of mental disease. On reading some treatises we might almost conclude that the nervous elements enjoy an immunity from disorder and disease, and that all evils that befall them are brought about through their supporting and ministering tissues. So completely indeed are the nervous elements sometimes ignored, that it would almost seem as if mental life was carried on without them, and as if they merely stood by consenting in an altogether superfluous sort of way. And yet these nervous elements are, as Rindfleisch has called them, the holy of holies, the all in all. They do not exist for membranes, vessels and fibres, but membranes, vessels and fibres exist for them; and of all the diseases to which the nervous system is liable, an immense majority probably originate in them. Of infinite complexity of structure, delicacy of organisation, and range of activity, they are readily thrown out of gear and then spread devastation around. 'For practical purposes,' says Mr. Simon, 'the state of the covering membranes of the nervous system may be regarded as a mere index of changes more or less distinctive, which their centres, in their own intimate composition, have at the same time undergone.'¹ And for practical purposes, therefore, the thickening and adhesion of the pia mater in general paralysis, may be regarded as an index of changes in the cortex beneath. That this is so will, I believe, be shown before long, by a demonstration that these adhesions are often distributed, not by continuity of structure, nor community of blood supply, but by a law of functional connection.

¹ 'A System of Medicine,' edited by J. Russell Reynolds, M.D., article, 'Epidemic, Cerebro-spinal Meningitis,' vol. ii. p. 921. London, 1868.

If it should be shown that lesions exist in two parts of the cerebrum which are remote from each other, and which have not the same vascular service, but which are frequently associated in action, then an additional and strong reason would be afforded for holding that it is from a change in the activity of the nerve elements that the disease springs. In the meantime, however, it is tolerably evident that the adhesions in general paralysis are not vascular in their origin and character, and that the disease does not, as has been ably maintained by Dr. John P. Gray and others, begin in the adventitious coat of the arterioles and large capillaries, for in that case we should expect to find these adhesions in the depths of the sulci, where the most numerous vessels enter, and not on the summits of the gyri, where the vessels are comparatively sparse.

Now, no explanation has yet been offered of the fact that adhesions of the pia mater are limited to the summits of the gyri, as they undoubtedly are, never being found extending down their sides nor at the bottom of the sulci, and it is desirable to consider this point. We are not, as yet, justified in saying that there is any structural or functional differentiation of the cortex at the top of a gyrus from that on its side. At the very bottom of a sulcus where the fold occurs, there is some compression of the cortex and dislocation of its elements, and it might be supposed that there was complementary stretching of the cortex over the top of a gyrus, but we have no evidence of this, and even if we had, it would not help us to account for the occurrence of adhesions in this situation. The changes in the nerve cells and fibrils which have been microscopically demonstrated in general paralysis, are not confined to the summits of the gyri, but affect all parts of their superficies; and if the primary morbid process in general paralysis is localised in these, it is clear that it is not to that primary morbid process that the adhesions of the pia mater are to be attributed. Then the thickening and œdema of the pia mater, resulting from the protracted and often recurring hyperæmia, induced by the morbid nerve process in general paralysis, is not confined to the surface of the cerebrum, but is as decidedly present in the duplicatures which sink into the fissures as in the folds which cover the

convolutions ; and it is clear, therefore, that it is not to the morbid changes in this membrane, nor to those in combination with the changes in the nerve tissue, that the adhesions are to be traced. Some other factor must have been at work in their production, and that other factor is, I believe, to be found in the mechanical contact of the summits of the gyri with the hard cranial walls. The living brain is, by virtue of its active blood circulation, in incessant molecular movement, and it is by no means proved that cardiac and respiratory variations in the encephalic vascular areas do not take place, that there is not an ebb and flow movement of the spinal fluid, and that the two surfaces of the arachnoid, or, if it is preferred, the arachnoid and epithelial lining of the dura mater, do not, to some extent, glide backwards and forwards on each other. The brain shows vigorous respiratory pulsations when the skull is opened, and a manometer communicating with the interior of the cranium, exhibits respiratory and cardiac oscillations; and it is not improbable that there is some degree of movement, even when the skull remains entire. But at any rate great variations must occur in intracranial pressure. When the circulation in the brain is active or when hyperæmia exists, the pressure exerted upon the convolutions becomes considerable, as may be seen in the brains of those who die when the organ is hyperæmic. Thus in some forms of acute and epileptic mania, the gyri are found flattened, and the openings of the sulci reduced to scarcely perceptible lines, just as in cases of cerebral tumour, or hydrocephalus internus. Then, whenever there is active turgescence of the brain, there must be a proportionate diminution of the cerebro-spinal fluid, and from this it results that the surface of the arachnoid is drier, less efficiently lubricated, and more liable to be detrimentally affected by friction or pressure. The dryness of the arachnoid will be greatest where the hyperæmia is most intense, and there the pressure upon the bony cranium will be most severe, just at the very point where it is likely also to be most pernicious. For healthy brain and its membranes may, we know, sustain pressure, even of a severe character, without undergoing inflammatory change, but

with irritated brain tissue and hyperæmic membranes the case is very different. Here we should expect an increase of irritation, a further advance in the inflammatory process, and an exudation, such as we have already alluded to, which may readily become a cementing medium between the pia mater and the cortex.

And there is a special reason why an exudation in this position should become a cementing medium and set up adhesions. It is an established fact that inflammatory exudations are powerfully influenced as to the transformations they undergo by the character of the tissue into which they are infiltrated, and that an exudation finding its way into connective tissue is apt itself to become connective tissue, being aided in that metamorphosis by the fibres amongst which it has intruded. The fibres, as Billroth has shown, are not merely pushed asunder but undergo softening and fusion, and, with the exudation, become converted into a delicate network resembling newly developed cellular membrane, and pass into connective tissue. Well, the outer layer of the cortex which is in contact with the pia mater is said to belong to the connective tissue system. The intercellular matrix of this layer is composed of a meshwork of minute fibrils which are largely derived from corpuscles. In this layer are seen the connective tissue corpuscles of Deiter, which have such numerous connections with each other and with the vessels, and in it also there are round or oval corpuscles containing a few small granules, and other corpuscles, which have two or three branches which anastomose with the branches of similar bodies in the pia mater. I believe I am correct in stating that the connections thus formed between the pia mater and the first layer of the cortex are most numerous at the summits of the gyri. These connections are, of course, very likely to become lines of propagation of pathological changes from their one point of attachment to the other, and centres for the multiplication of connective tissue.

I have said that we are not as yet justified in alleging that there is any difference of function in the different parts of a convolution. It would not, however, be unreasonable

to suppose that this may be the case. The convolutions which are continuous with one another, which are indeed mere folds in the cerebrum, have, it has now been proved, differentiated functions. That sharp lines of demarcation separate different functional areas, is, to say the least, improbable. More likely is it that there is a gradual transition from one species of functional activity to another, and if that be so, the relations of the grey matter at the bottom of a sulcus may be altogether different from those of the grey matter on the ridges of the neighbouring gyri. Then, again, it seems to me contrary to all analogy and to the facts of evolution to suppose that all the cells of the cerebral cortex are developed and reach maturity simultaneously. A study of comparative anatomy suggests that the six different cortical layers are developed not simultaneously, but consecutively, and just as there are centres of ossification in bone, so may there be centres of cerebration in brain. There may be certain points on the cerebral surface in which the cells first grow, put forth processes, and form connections, and from which cell-growth and development gradually extend around. In that case certain points of the cerebrum, perhaps certain points in each gyrus, might, as the parts finally evolved, have a primary proclivity to dissolution, which takes place in the inverse order of evolution, or might, as the seats of habitual activity, be most liable to suffer from irritation and hyperæmia.

The explanation now suggested of the cause of the limitation of the adhesions of the pia mater to the summits of the gyri, enables us if adopted to understand how it is that in certain cases of general paralysis these adhesions may be wanting. In these cases the disease is of the slow degenerative type. There has been wasting of the brain before any active hyperæmia sets in, so that when the latter does supervene, if it comes at all, the surface of the cerebrum is bathed in serum, and there is not that friction or pressure against the skull which has been supposed to be instrumental in increasing the irritation and so leading up to the adhesions.

Whether or not the inflammatory process in the cineritious substance, which seems to be most active in the summits of the gyri, dips into the medullary matter, whether or not a neuritis ultimately pervades the whole nervous system.

we are not in a position to decide. No doubt widely diffused changes have been found in advanced cases of the disease, and some of these look as if they were essentially connected with it. This is so with the neuritis and atrophy of the optic nerve, so well described by Dr. Clifford Allbutt.¹ But other changes, again, seem to be consecutive in character, and to depend upon the disablement which the disease entails. In this category must be placed the fatty degeneration of the muscles that has been noticed by Dr. Ashe,² and the fasciculate atrophy of nerve tubuli occurring in the nerve trunks of the limbs, investigated by Dr. Bevan Lewis.³ Muscles and nerves always dwindle and degenerate if disused or very little used, and it would indeed be wonderful if they did not do so in a helpless and bed-ridden general paralytic, who is in a state of marasmus. For the rule is that towards the close of the disease nutrition is very imperfectly carried on, and general emaciation and decay set in. Then it is that bedsores become troublesome. But general paralysis is essentially a cerebral disease, with mental and motor symptoms, and it is in the cortical substance of the cerebrum that its fundamental conditions must be sought.

I have now endeavoured to show, 1st. that the adhesions of the pia mater to the grey matter of the brain are the most frequent and characteristic of the pathological appearances found in general paralysis of the insane; 2nd. that they are caused by a chronic adhesive inflammatory process, springing out of excessive functional irritation, and proceeding to disintegration of the cerebral grey matter; and 3rd. that, speaking generally, they represent the course and distribution of the morbid processes in which the disease essentially consists. If these propositions should be established, or even if there should be a probability of their correctness, the adhesions in general paralysis assume an importance that can scarcely be exaggerated and merit the closest study. These adhesions would then become the legible records of a

¹ 'On the Use of the Ophthalmoscope in Diseases of the Nervous System, and of the Kidneys,' by Thomas Clifford Allbutt, M.D., London, 1871, p. 193.

² 'Some Observations on General Paralysis,' by Isaac Ashe, M.D. 'Journal of Mental Science,' April, 1876, p. 82.

³ 'On the Histology of the Great Sciatic Nerve,' by W. Bevan Lewis. 'West Riding Lunatic Asylum Medical Reports,' vol. v. p. 85. London, 1876.

series of experiments of the most instructive kind performed upon the human brain by disease. They would mark upon the surface of the cerebrum, the centres, from stimulation and destruction of which, by a morbid process, a certain series of symptoms has proceeded. They would enable us to trace to their source, with more or less precision, the psychical and motor disorders and impairments of general paralysis, and they would thus guide us to the localisation of the functions of the brain. Thanks to the general indications of Hitzig, the precise work of Ferrier, and the clinical observations of Hughlings Jackson, we are already in possession of invaluable information upon this subject, and fortunately, what we already know may be applied as a test to the trustworthiness of the guide proposed to conduct us to fresh discoveries. There are, in general paralysis, invariably certain motor symptoms, involving, in different degrees and in various orders of succession, the muscles of the face, tongue, hand, arm, leg, etc. There are in the human brain certain areas which, by arguments from homology as well as by clinical facts, can be proved to be centres of representation of the movements of the face, tongue, hand, arm, leg, etc. Do adhesions occur over these areas in cases of death from general paralysis? Yes, almost invariably,—a fact which in itself gives plausibility to the claim advanced for these adhesions to be regarded as reliable *vestigia* of the diseased action.

The nature of the morbid process in general paralysis as it has been here described is such that in its course, it imitates, as it were, the experiments of the physiologist upon the brain. In travelling over the hemispheres it first stimulates and then breaks up a centre or centres; it first produces a discharging and then a destroying lesion. It reveals to us the effects, 1st. of comparatively slow, feeble, incontinent discharge of certain tracts and areas of grey matter; 2nd. of the sudden, excessive, and explosive discharge of wider tracts and areas; and 3rd. of the gradual loss of functional activity of the areas and tracts which have been so discharged. It thus affords peculiar facilities for ascertaining in which regions of the cerebrum certain groups of movements are most represented, and in what order these

movements are grouped and arranged. True, the irregular manner in which the symptoms are manifested, the complexity of the phenomena to be observed, and the indefiniteness of the *post-mortem* appearances, must, in a great number of cases, make the analysis of the symptoms and the connection of particular symptoms with particular lesions, an eminently difficult, and sometimes an unattemptable task. But in some cases the symptoms are simpler and the appearances more definite, while in most cases some general relations may be traced out. And each case analysed, however imperfectly, will afford a little vantage ground from which to approach the next case, so that obstacles will melt away before laborious research.

In order that full use may be made of the adhesions in general paralysis as guides to the progress of the disease, and to an enlarged knowledge of cerebral physiology and pathology, two things are requisite—first, an accurate record of the symptoms, and secondly, an accurate chart of the lesions. By a comparison of these we can alone arrive at the knowledge which we desiderate.

The first requisite—a minute and accurate report of symptoms—may be easily got by a little watchfulness and work, and the second requisite—a correct chart of the lesions—is also readily attainable by a process which has been recently adopted at the West Riding Asylum. Having long believed in the significance of the adhesions of the pia mater in general paralysis, and having been especially impressed by the importance of tracing them out, since I witnessed Ferrier's masterly experiments, I have made many attempts to do so, but until lately without much success. The brain in general paralysis is so soft that stripping with the forceps is not very successful. Flakes of grey matter which are not adherent are apt to be torn away with those that are. The isthmuses and strands that lie between the adherent patches, are exceedingly liable to be torn through by the tough and thickened vessels, and a mere touch of the forceps' points in pinching up the pia mater will produce the semblance of an adhesion where none exists. Then, if the stripping is conducted with the brain under water, the

softening increases, and portions of grey matter may be washed away. With extreme care and nicety, a brain in which there are numerous adhesions may be stripped with the forceps in a satisfactory manner, but as a rule, the results obtained are not encouraging. It was when visiting Oxford and looking at some brains preserved by Professor Rolleston, that a better method of obtaining a view of the adhesions in general paralysis occurred to me. These brains were preserved in nitric acid, which has the property of hardening and condensing the cerebral substance, and which at the same time blackens and eats away all animal membranes with which it comes in contact. It struck me that if the brain of a general paralytic were treated in this way, the pia mater would be consumed, and the convolutions left entire and bearing the marks of the adhesions. The experiment was tried, and with gratifying success. It was found that by steeping the brain for a few weeks in a mixture of one part of strong nitric acid to eight or ten of water, the membranes were completely removed, while spots where adhesions had existed were left rough, eroded, and readily recognisable. Dr. Bevan Lewis, my former colleague, who has now prepared a number of brains in this way, and to whom I am indebted for much valuable assistance, has found that it is better not to wait for the slow destruction of the pia mater by the acid, but to peel it off whenever it has become black and the brain substance hard. It then separates with great facility, and leaves the abraded areas so distinctly marked out that no difficulty is experienced in making drawings of them, or in marking their position upon diagrammatic outlines of the cerebral convolutions.

In the six following cases of general paralysis, drawings of the brains, which have all been prepared in nitric acid by Dr. Bevan Lewis, have been made in two instances by that gentleman, in two by Mr. Crochley Clapham, and in two by myself. These drawings, which have been selected out of a series of drawings of twelve brains now in my possession, will convey a general idea of the various ways in which the adhesions are distributed in cases in which they are numerous and in cases in which they are few in number. In

reading the notes of these cases which it has been thought well to publish along with the drawings, it must be remembered that they were not taken with a view to publication, nor to the elucidation of the pathology of general paralysis, nor indeed with a view to any special enquiry. They are merely rough notes taken in the ordinary way in cases which presented no special feature of interest at the time. Had it been contemplated that tracings would be obtained of the lesions in these cases, their psychical and motor symptoms would of course have been set down with much more fulness and minuteness.

CASE I.—J. T. Occupation, mason. Admitted on October 3, 1873.

Facts observed by medical man.—This man was acquitted on his trial for larceny on the ground of insanity, but during his stay in prison he is said to have shown no symptoms of insanity.

Age 36. Married. Religious persuasion, Church of England. Previous place of abode, Bradford. Not epileptic.

Has 2 children. Can read and write.

History.—(From the Prison Hospital warder, and from the certificate of Dr. Wood.) On August 21 patient was tried at the Wakefield Sessions for stealing 7 Pigs, but was acquitted on the ground of insanity, and ordered to be detained during Her Majesty's pleasure. Since that time he has been in the hospital of the prison, where he has been quiet, rational and well-behaved. He was acquitted at the trial on account of a doctor having come forward and stated that he was insane. He is not known to have been in an asylum. Nothing further known about him.

State on Admission. Mental Condition.—He is a little slow in answering questions; his memory is clear. There is some depression connected with money matters. Since last Christmas, according to his own account, he has had to pay 10s. a week to make up a deficiency. He has had no sleep at night, and has had a gradual diminution of muscular strength. He has fallen off ladders and platforms whilst at his work. The quality of his work deteriorated, and his masters complained. He has no doubt that he is able to return to his work, and that he is fit for it, although his hands are very tremulous and shaky. He thinks he will make 'a bit of brass,' and he says he has been sober until this trouble. He once had an injury to his head, and was unconscious for 2 hours. This was when an apprentice. His family are healthy. No family history of insanity. He has had dizzy feelings in his head. His sight and hearing are good. No numbness nor formation of extremities. He is certain that he is getting better.

Physical Condition.—Circulatory System.—Heart systole is rough, and blowing at apex.

Respiratory System.—Normal.

Digestive System.—Normal.

General Appearance.—Height, 5 ft. 7. in., weight, 144 lbs. Complexion

florid. Expression of countenance stolid, and when speaking there is some tremor of the muscles of the lip. Tongue very shaky, and voice tremulous. There is an occasional impediment in his speech. Pupils slightly contracted and a little irregular at the margins. Fairly but not well nourished. Unsteady and muscular twitchings all over body.

Progress of Case. October 4.—R. Ext. Physostig., gr. $\frac{1}{4}$ ter in die.

October 14.—The patient thinks his tongue 'has gone up into his head,' and that it is fixed there.

April 10, 1874.—Symptoms of general paralysis almost arrested. Mind quite clear and free from delusions. The only remaining symptom is a slight tremulousness of the facial muscles, and a certain hesitation in articulation.

October 14.—Has got much worse during the last few months. Much more demented. Seems to have no idea of the season, but thinks it is the beginning of the year. Articulation is becoming very much embarrassed, and movements generally clumsy and uncertain. He is still able to work with the masons.

January 18, 1875.—Patient slowly but surely gets worse, although he still goes out to work with the masons, and is quiet and docile.

June 2.—He was observed to be more shaky than usual this morning, and there seems a slight loss of power in the right hand. He has not taken food so well for a day or two, and appears to be losing flesh. Pupils are irregular, the right larger than the left. Articulation gets more and more involved and indistinct, and he is very much demented; at times dirty in his habits.

June 22.—Much excited, and losing flesh rapidly. Physical symptoms making rapid progress. Ext. Physostig., which had been omitted for some time, to be resumed.

September 14.—Getting very feeble; is now unable to move about. Is very restless and noisy at times. Has been suffering from boils, which are now better. Has also a sore commencing over the sacrum.

September 22.—He has been very quiet for the last few days; lies in bed perfectly still, with his eyes shut, and refuses to speak; is taking food well. His sores are improving.

September 29.—He has been excited during the night. This morning has twitchings, especially of right arm.

October 6.—Since last attack of excitement has undergone no mental change, and lies in a dull, lethargic state. When questioned, appears to understand what is said, but makes no effort to answer. Takes his food well with feeding. Sleeps well. No twitchings.

October 7.—Still in a lethargic state. To-day has trembling convulsions. The limbs affected are the right arm and leg.

October 18.—Last night he was again somewhat excited and convulsed. Thirty grains of chloral were administered, and afterwards he was quiet. This morning the pulse is quick, but he is quiet.

November 9.—Is still very weak, but somewhat better.

November 29.—Had a convulsive attack last night, in which the right arm was affected. This lasted about four hours. To-day there are no convulsive movements of any part of the body, excepting the right arm, which

twitches slightly. Pulse 150. Breathing laboured, and inclined to be stertorous.

November 30.—Seems better this morning. The pulse is quieter, but very feeble. There are no muscular tremors.

December 1.—Last night the breathing became much quickened, and his pulse very quick and weak. He died this morning.

Cause of Death.—General paralysis of the insane. Suppurative disease of kidneys.

P. M. 77 hours after death. Rigidity present in legs. Body greatly emaciated. Blue colour of skin over abdomen. Small sore over left trochanter. No fat over abdomen. There are discolouration and abrasions where the skin has been pressed, viz. over the ankles, elbows, &c. The foreskin is excoriated. There is hypostatic congestion.

Head.—Of average thickness, fair capacity, and symmetrical. The bones are pale. *Dura Mater.*—The sinuses contain dark clots. The arachnoid is exceedingly white and opaque over the frontal and parietal lobes.

The *Pia Mater* is tough, and adherent to the grey matter. Convolutions in frontal and parietal lobes greatly wasted, and the sulci water-logged. Brain is generally anæmic. It is preserved in nitric acid. $\frac{3}{4}$ iij. of fluid escaped from the surface and interior of the brain. The whole brain weighs 1,285 grammes.

Thorax.—Heart weighs 298 gms. No fluid in pericardium. On the left side there was a membranous clot. The valves are competent. The muscular substance is thin.

Lungs, right, 790 gms., is generally adherent by a tough fibrous membrane, and there is one large black carbonaceous mass the size of a hen's egg.

There are some tuberculous nodules at the apex, and the lower lobe is cedematous. Left lung weighs 645 gms., and is in much the same condition.

Abdomen.—Liver weighs 1,235 gms., is pale, soft, and fat. It is mottled externally.

Kidneys.—Right weighs 215 gms. On its outer surface is an abscess posteriorly, containing several ounces of purulent fluid, which escaped during section; the lower $\frac{3}{4}$ of the kidney is found to be infiltrated with pus, and the proper substance of the kidney is no longer distinguishable through numerous small abscesses which pervade it.

Left Kidney weighs 170 gms. On the posterior surface is a projecting abscess the size of a large walnut.

Spleen weighs 113 gms.; pale and tough.

CASE II.—W. R. Occupation, tailor. From Leeds. Admitted on March 30, 1874.

Facts observed by medical man.—Looks wild and excited. Talks in a hesitating manner, as if unable to give expression to his ideas. When questioned, is unable to keep his attention fixed to one subject, but answers in a rambling, incoherent strain. Memory defective.

Other facts communicated.—Frequently breaks out in the most violent manner, under the delusion that he is about to be murdered. Has been kept in a padded room for the last 2 days. Age 33. Married.

Religious Persuasion.—Church of England.

Insane about 3 days.

Supposed cause not known. Not known if epileptic. Not suicidal. Is dangerous to others. Has 2 children. Can read and write. Is attentive to the calls of nature.

History from Relieving Officer.—He was taken to Leeds Infirmary on March 25. After being there about 24 hours, he suddenly became very excited, taking hold of the poker, rushing downstairs, and trying to strike whoever came in his way. Since then he has been confined in the padded room, and has been very restless. It is not known for what the patient was taken to the Infirmary, for nothing is known of his previous history or of his family, except that his wife lives at Skipton Road, High Wortley.

Mental Condition.—It is almost impossible to fix his attention upon anything, and even when he can be induced to answer questions, his answers are absurd and contradictory. At one time he says he has been here 6 months, and again 2 years. At present his attention seems entirely engrossed by some imaginary thing in his hands. When asked what it is, he says it is some dirty thing. He is in the habit of picking out his eyelashes. Can give no reliable account of himself. Knows he was brought up at the Industrial School at Leeds, and has been a tailor by trade. He is somewhat emotional. When asked if he is happy, begins to cry, and says he would as soon die. Expression heavy; articulation slow and hesitating.

Physical Condition.—*Circulatory System.*—Apex-beat of heart not definable. Sounds feeble. No murmur. Second sound accentuated at base.

Respiratory System.—Normal.

Digestive System.—Bowels confined.

General Appearance.—Height, 5 ft. 1 in. Weight, 130 lbs. Hair, black. Eyes, brown. Pupils equal, but sluggish. When asked to put out his tongue, it is protruded with a jerky movement, and drawn in directly. When asked to open his mouth he cannot always do so. There is great tremor of the muscles of the face. Is in good bodily condition. Is rather unsteady in his movements, but there is no difference in the muscular power on the two sides. The right eye is inflamed, apparently from irritation caused by his pulling out the eyelashes.

Diagnosis.—General paralysis.

Prognosis.—Unfavourable.

Treatment of Ext. Physostig., gr. $\frac{1}{4}$ ter in die.

Progress of Case. April 17.—Is still restless, but less so than when admitted. Occasionally has a tendency to throw off his clothes, and during the night gets out of bed, and tosses his bedclothes about. He has a frightened look, and when spoken to, shrinks away, saying he is in prison, and seems as if he feared some injury. Takes food well. Thickness of speech and change of voice.

April 29.—Patient's wife, who visited him to-day, states that in May last he had a sunstroke, and after that during the summer he had repeated attacks, which she calls fits, in which his limbs remained rigidly fixed and straight, and his hands clenched for a time. After each attack he remained for a few days unable to speak, and when he recovered complained of pain at the back of the head and dimness of sight. He has always been a sober man. Has

been married about ten years, and has had ten children; six were premature, and two others died in early infancy; only two alive. Nothing known of his relations.

May 1.—His brother visited him to-day, and states that the family consists of 5 daughters and 3 sons, of whom W. is the youngest. The others all healthy. Father and mother died at a good age. He knows of no insanity in the family.

May 4.—Getting rapidly worse. To-day he is quieter, but looks languid and feeble. Has not taken food so well as usual, and does not speak. Ordered to bed.

July 8.—Has improved considerably in bodily condition. Has made several attempts to strangle himself with his scarf lately. He makes sudden attacks upon other patients, and is very violent at times, especially during meals. Rubs his head, and complains of pain there. The fits of excitement last a few minutes only, he soon exhausts himself, and settles down.

July 11.—Again made a determined attempt to strangle himself, and expressed his wish to be out of the world. Is unsteady in gait.

September 25.—Has been much quieter of late, and has improved greatly in bodily condition. There has been no suicidal tendency manifested for some time. Has delusions of an exalted nature, e.g. that his bowels are full of diamonds, which he passes in his stools. He is considerably demented, but is still able to do a little work as a tailor if watched and directed.

October 12.—No change since last report. Has shown no suicidal tendency since removal to No. 2. Still employed in tailor's shop.

January 30, 1875.—General paralysis steadily on the increase, but he is not quite so depressed.

June 10.—Symptoms progressing slowly. Has become very demented, and is not able to do any work. Shaky in all his movements.

June 23.—Had a convulsive seizure affecting the left side lasting three minutes. His bowels were well opened with castor oil this morning.

September 22.—Little change has occurred since last report. He is very demented, and shaky in his muscular movements.

September 27.—He had a convulsive seizure last night. The left foot twitched, and he was quite helpless. The convulsions have ceased now, and he is not paralysed.

September 29.—The fits have been continuing since last report. Last night he had 31 convulsive seizures, and this morning 10, the last 2 in presence of medical officers. During fit the head was first turned to left side, and this was not accompanied nor preceded by any exclamation. The muscles of the face were then convulsed, the twitchings on the left side being more powerful than on the right. The eyeballs were turned downwards. The arms were then tossed first in a vertical direction, and then, having been placed across the chest, were thrust laterally towards each other. The legs were drawn upwards, and the thighs semiflexed on the abdomen. While in this position they were strongly convulsed. Succeeding upon this there were tonic spasms, in which he again turned completely on his back. The whole seizure lasted about half a minute. After an interval of a quarter of a minute the convulsions again commenced, with no difference from the last. He then fell into a heavy condition, with slight stertor of breathing, as before

the attack. The pupils were somewhat contracted, but equal. Reflex action more than usually acute, both with regard to tickling and pain all over the body and face. Has taken very little food. An injection was ordered to move the bowels, and the following injection to be used three times a day:—Port, $\frac{3}{4}$ iv.; beef-tea, $\frac{3}{4}$ v., 1 egg; 15 grs. of hydrate of chloral.

October 1.—At 8.15 yesterday morning was found to be breathing with great rapidity, and with marked stertor; 51 respirations per minute. He was convulsed about every 5 minutes, but the convulsion did not affect one side more than another. Sometimes they commenced on the right and at other times on the left side. The pupils were unequal, the left being the largest. Pulse 150. The temperature, which in the previous night had been very high, was reduced to a little above normal. From the commencement to end of seizure chloral did not have the slightest effect in warding off the convulsions. The patient gradually sank, and died at 11.15.

Cause of Death.—General paralysis of the insane.

Died September 30, 1875, at 11.15 A.M.

Examination 49 hours after death. Weather warm. The body is plump and well nourished, a thick layer of fat covering the abdomen. Rigor mortis is present, and there is a small amount of hypostatic congestion.

Head.—The skull is of a round shape, and of dense consistence. It is symmetrical, and the bones composing it have a bluish tinge owing to venous engorgement. The dura mater is thickened but not adherent, and its sinuses contain clots and dark fluid blood. The arachnoid has a greyish, cloudy aspect over the frontal and parietal lobes, and is floated up by a large quantity of subjacent serous fluid. The superficial veins are engorged with dark blood, and all the vessels of the pia mater are dilated. The aspect of the brain generally is that of intense venous congestion. The gyri are much wasted in the frontal and anterior parietal regions, and the brain substance is decidedly soft. The walls of the large arteries at the base are thick, but not atheromatous. There is no venous engorgement around the cerebellum. A considerable amount of serous fluid escaped from the ventricles. Whole brain, 1,293 gms. Other weights are not taken, as the brain is immersed in nitric acid, with a view to subsequent examination. Immediately on immersion in the acid the membrane became opaque and white, the blood in the vessels brown, and the brain substance harder.

Thorax.—No fluid in the pericardium.

Heart weighs 293 gms. Right side full of dark clotted blood. Left side empty. Valves normal. Substance soft and flabby. Left ventricle dilated.

Right lung, 581 gms., adherent in its upper lobe by old fibrous bands. Somewhat congested, but crepitant throughout. No tubercle.

Left lung, 496 gms., adherent at lower margin of upper lobe, also congested, but crepitant. No tubercle.

Abdomen.—Liver, 1,095 gms. Normal.

Spleen.—92 gms. Normal.

Right kidney 110 gms. Left kidney 110 gms.

Capsules of both slightly adherent at a few scattered points.

Cortical substance pale. Fibrous tissue in excess.

Bowels not loaded.

CASE III.—F. S., Occupation, pianoforte-tuner and teacher of music. Chargeable to Hunslet. Admitted on April 16, 1875.

Facts observed by Medical Man.—Demented appearance. Desires to ramble hither and thither with no fixed purpose. Shows symptoms of general paralysis.

Age, 40 years. Married. Religious persuasion, Church of England. Previous place of abode, Union Workhouse, Hunslet. First attack. Insane about 10 months.

Supposed cause, over-exertion and slight sun-stroke. Not epileptic. Not suicidal. Not dangerous to others.

Has no children. Can read and write. No relatives similarly affected. Does not tear clothes, nor break windows. Is attentive to the calls of nature.

As a professional cricketer he has been struck by the ball occasionally.

History.—This is patient's first attack, and is said to have existed for ten months. Besides his ordinary occupation he is a professional cricketer. It is stated that he has a tendency to wander hither and thither. He has shown great deficiency of motor power. Has spoken about going to Harrogate to stay for several months, which his position cannot admit of. During the last ten months he has lost his business through incapacity; he became bankrupt, and had to go to the workhouse. During his residence there he had large ideas about travelling long distances, but not about being possessed of large sums of money. He shows exalted ideas of power. Is said to have had slight sunstroke while cricketing, and to have been often struck by the ball. There is no family history of insanity. He has had no children. The relieving officer states that he has lived a rather fast life with regard both to drink and women.

Mental Condition.—He understands what is said, and answers some questions correctly regarding the early part of his life. He gives confused and contradictory statements regarding its later period. He thinks that he is in Leeds Workhouse, that he came here yesterday, and then went back to Leeds. Is unconscious of any deterioration in his health; says he is as strong and fit for his work as he ever was. He has a very pleased and beaming expression of face. Does not seem to have any exalted ideas of wealth or grandeur at present. His articulation is very much involved and hesitating.

Physical Condition.—Height, 5 ft. 4 in. Weight, 150 lbs. Hair, light brown, very thin on the crown. Eyes, bluish-grey; left pupil a little larger than right one; both are active. Complexion, sallow. He is in good condition; there is a slight bruise on leg above left knee, also a large superficial cicatrix on chest, said to have been the result of a burn. Is extremely tottering in his gait. The grasping power of both hands is very feeble and 'jerkily' exerted. Heart and lungs are normal. Pulse 96. Has taken his food. Urine, sp. gr. 1,010, and normal.

Diagnosis.—General paralysis.

Prognosis.—Unfavourable.

Treatment.—Iodide of potassium.

Progress of Case. May.—Much the same as on admission. Has a smiling and benevolent expression, thinks he is stronger than he ever was, and is going to Scotland to play in a cricket match. Movements very shaky and embarrassed.

June 7.—Is hardly able to get up and down stairs. His articulation is very much affected.

June 17.—Yesterday at dinner-time he had a sort of fainting-fit, lost the use of his legs, and vomited his dinner. Had a dose of castor oil, and his bowels were well opened during the night. To-day he is still feeble, and not able to walk about; he looks drowsy, and does not take notice. Keeps his left arm rigidly fixed in the flexed position. The head and eyes have a tendency to roll to the right, and the left pupil is considerably larger than the right one. He is conscious; tongue white; pulse about 90, and rather feeble; breathing is natural. Ordered Mist. Gentianæ.

June 23.—Ordered Potass Iodid., and Bichloride of Mercury.

September 21.—He is rapidly going downhill. K. I. has had no beneficial effect. He has been in bed for some time with an ulcer on his left leg.

September 22.—Can make out No. 1 Jaeger text-type at a distance.

October 14.—Has Herpes Zoster on the left side over the lumbar region. Nitrate of silver lotion applied.

November 29.—Has been suffering for some days from diarrhoea. The motions are slimy and mixed with blood. Ordered 3 ss. doses of Pernitrate of Iron.

December 1.—Though the diarrhoea has been controlled, he has been losing ground. Last night his breathing was much quickened, and his temperature and pulsations increased. He died this morning.

Cause of Death.—General paralysis of the insane. Pulmonary congestion.

Died at 12.35 A.M. on December 1, 1875.

P. M. 41 hours after death. Weather cold and wet. Body somewhat emaciated, but there is a layer of fat on the abdomen. There is lividity and excoriation of the scrotum, due to the dribbling of urine. Rigor mortis is present in both the legs and arms, and there is a slight degree of hypostatic lividity and congestion.

Head.—Skull of average thickness and fair symmetry. The bones are pale.

Dura mater, not thick nor adherent.

The sinuses contain a small quantity of dark fluid blood.

Brain is of a generally shrunken and wasted appearance.

The arachnoid is thickened and cloudy over the frontal and parietal lobes.

There is a large amount of subjacent serum.

The convolutions of these lobes are much wasted and the sulci enlarged.

Pia mater is adherent to the summits of the gyri, and the cerebral substance is soft.

Ventricles contain a large amount of clear serum.

Vessels at the base not atheromatous.

The whole brain weighs 1,099 gms. It is preserved in nitric acid solution.

Thorax.—Heart.—Pericardium free from fluid. It weighs 247 gms. Its cavities are empty.

The mitral orifice is of average size, but there is no deposit on the mitral flaps. The valves are competent.

Lungs.—Right, firmly adherent by old fibrous tissue. Weight, 492 gms. It is sub-crepitant throughout, and contains no tubercle. Left, adherent and in the same condition as the other. Weight, 375 gms.

Liver.—Weight, 1,230 gms. Normal.

Spleen.—Weight, 121 gms. Normal.

Kidneys.—Right, 115 gms. Left, 120 gms. Normal.

Capsules free.

Intestines.—No ulceration.

CASE IV.—B. W., a slop-dyer, Halifax. Admitted December 29, 1873. *Æt.* 36. Married. No known cause.

Facts observed by medical man.—Answers questions at random, without any bearing on the subject. Says he is expecting to see his wife, who died 10 months ago.

Brother died insane 12 years ago.

History.—No information could be obtained from the relieving officer, who came with the patient. From the form of admission it is gathered that it is his first attack, and commenced about seven months ago; that he raves on same subject, and is alternately violent and depressed, and is dirty in his habits; that he wanders about the streets, takes things out of his house, and keeps ordering things to be sent in indiscriminately. He constantly threatens to commit suicide.

Mental State.—Since admission he has been quiet, and has slept well. He has taken food fairly. He understands what is said to him, and gives apparently an accurate account of most of his past life. States that he has been married 20 years, has no family, and has been separated from his wife for the last 7 years. He says he has a brother and sister alive, and remembers a brother dying in the Asylum. Says he has plenty of money in some co-operative society, and draws 20*l.* a day from some lodge. He acknowledges having ordered lots of goods, and can give no reason for doing so, except that he wanted them. His expression is that of a general paralytic.

Physical Condition.—Pulse 104, compressible and weak. Heart sounds are feeble.

Respiratory System.—Normal.

Digestive System.—Tongue clean and dry. Has voided his urine in bed.

Height, 5 ft. 6 in. Weight, 130 lbs. Hair, light brown. Eyes, grey-blue; pupils unequal, the right being considerably the larger of the two.

Diagnosis.—General paralysis.

Prognosis.—Unfavourable.

Treatment.—Ext. Physostig. $\frac{1}{4}$ ter in die.

January 8, 1874.—On examination of chest there are no decided indications of present disease, but the respiratory murmur is harsh at several points, and vocal resonance somewhat increased at the left apex. Patient states that he coughs some phlegm. Ordered Tinct. Ferri Mur. m. xv. and Ol. Morrhæ $\frac{3}{4}$ ter in die.

January 28, 1874.—Looks pinched and feeble. Ordered 4 oz. port per diem. He was put to bed a few days ago, as he appeared very feeble. It is found that he has necrosis in ball of great toe. Appetite poor, and does not take anything but liquid food. Poultices are ordered to great toe.

February 14.—Purged twice this morning. He is very feeble. Ordered brandy, $\frac{3}{4}$ viij. per diem, beef-tea, &c.

April 3.—Yesterday he had great toe amputated. The wound is healing well. Patient has fortunately been quieter than usual, and allows dressing to remain.

April 15.—Medicine stopped.

October 12.—Has improved in general bodily conditions, sometimes excitable and noisy, and very pugnacious at times. Has a habit of picking his nose, which he has disfigured very much, a considerable part of the septum being gone.

January 16, 1875.—He has allowed the nose to heal. The last report still holds good. He is very easily excited, uses the most fearful language, cursing and threatening those who annoy him. He is very fond of singing to himself in a high falsetto voice. Bodily health moderate; considerable anæmia.

June 8.—Symptoms have remained almost stationary since last report. His general bodily health is pretty good, and he is not so pugnacious as formerly.

June 13.—Has been getting considerably feebler the last few weeks, and often wet and dirty in his habits. To-day he has been purged three times; looks pale and ill. Pulse 108, feeble. Complains of pain in his bowels.

June 16.—Has had no diarrhœa for two days, and he wants to get up.

September 15.—Symptoms have made more rapid progress during last few weeks. Getting very shaky in his movements. Has begun to pick his nose again.

March 2, 1876.—He has been failing for a considerable time, and to-day he died from general paralysis.

Died March 2, 1876. Examination 39 hours after death. Weather mild. Body is considerably emaciated. No subcutaneous fat. Skin of scrotum excoriated, as from frequent wetting. Left knee much enlarged and deformed from some old injury, and the left leg is considerably swollen. Rigor mortis is present in a marked degree, but there is little or no hypostatic congestion. The skin of the abdomen is green from decay.

Head.—The skull is of average thickness, and rather soft. It is not quite symmetrical, but bulges a little posteriorly on the left side. The inner table has a slightly bluish tinge. The dura mater is normal, and the sinuses contain a small quantity of dark fluid blood. There is a milky whiteness of the arachnoid over the frontal and parietal lobes, the gyri of which are considerably wasted. There is no atheroma of vessels. The pia mater is tough, and adherent to the grey matter on the tips of the gyri.

Whole brain weighs 1140 grammes. It is preserved in nitric acid for subsequent examination. About 4 oz. of fluid escaped from the brain during its removal.

Thorax.—Pericardium empty. Heart weighs 235 gms. All the cavities contained organised clots. The valves are competent. On the aorta are numerous atheromatous patches.

Right lung, 763 gms. It is everywhere adherent by fibrinous membrane to wall of thorax. It is generally congested and œdematous.

Left lung, 486 gms. Congested and cedematous.

Abdomen.—Liver, 1,325 gms., pale.

Spleen, 129 gms., firm.

Kidneys.—Right, 138 gms. Left, 138 gms.

Capsules, thin, and somewhat adherent. Substance fairly normal in appearance.

CASE V.—G. S., Leeds. Admitted November 11, 1873. Aged 41. Married. Second attack.

Facts indicating Insanity.—Expression excited, manner strange and restless; talks rapidly and incoherently; answers questions with all kinds of absurd nonsense. Has delusions such as that he is engaged to fit up all the cathedrals in the country with hot-water pipes.

History.—Patient was discharged from the Asylum about August 25 last as a case of general paralysis—arrested, having been admitted October 12 previous. Nothing further is known about him at present, except that two days ago he was found at night in the streets of Leeds with his clothes all in rags and in a queer and excited state. He was destitute, and could give no proper account of himself, so was taken to the lock-up at the Leeds Town Hall. There he got worse, and has been very excited and destructive. He is full of grand and exalted ideas.

Present Mental State.—He is restless and much excited both night and day, and full of delusions of an exalted character. Says he is going to Newcastle, where he has four churches to furnish with heating apparatus; that he has taken a house six storeys high, and built a large pleasure-boat, &c. His memory seems tolerably good, and he says that for weeks before he came here he was unable to sleep at nights. Generally his mind is absorbed in delusions, about which he is constantly talking.

Physical State.—A muffled and indistinct murmur present with the second heart sound. Impulse ill-defined. Pulse, 76, good.

Respiratory System.—Lungs normal; voice tremulous; speech hesitating and halting.

Digestive System.—Tongue very tremulous; appetite good; bowels regular. Passes water freely.

General Appearance.—Height, 5 ft. 10½ in. Weight, 170 lbs. Hair light brown; irides grey; pupils equal, but contracted. He is in fair condition, and free from bruises or eruptions. Surface of body generally somewhat pale, and there is a reddish scar over the left shoulder-blade. No vaccination cicatrix. There is a good deal of general muscular tremor, more especially in the muscles of the face.

Progress of Case. April 29th, 1874.—The symptoms have been almost stationary since admission, and he has been constantly employed. He has not, however, taken his food so well for some time, and seems to be losing flesh.

September 7.—For some time he has been getting more restless, and last night had a serious attack of excitement, tearing bedclothes, &c. To-day he is excited, shouting about diamonds, and money, and tearing his clothes. To have ʒ ss. of castor oil.

October 27.—Is quieter and less restless than before.

January 16, 1875.—Patient is excited and restless at intervals, quieting down after a few days, seemingly on account of physical exhaustion. He is quiet at present. Disease has not made much progress of late, though his bodily condition is on the whole deteriorating.

April 14.—Is getting very feeble, and going down-hill. Ext. Physostigma.

June 16.—Is getting varicose ulcers on legs. Is very weak generally; pulse weak. Ordered stimulants.

September 22.—He has laid on flesh since last note; he is exceedingly tottering in his gait and dirty. He can read No. 2 Jaeger—even No. 1 Jaeger text-type, with some difficulty—without the aid of spectacles at 10 inches' distance.

October 1.—Had convulsive attacks yesterday; given an injection. Is better to-day.

January 9, 1876.—Has had convulsive attacks recently, and has been getting weaker. He has also suffered through the existence of an impermiable stricture. He died this morning. Cause of death, general paralysis.

Died January 9, 1876. *Sectio cadaveris* 34 hours after death. Weather cold and damp. Body fairly nourished. There are marks of old ulcers and several small abrasions and pimples on the legs. Scrotum and penis swollen. Rigor mortis is present in both legs and arms. No hypostatic congestion.

Head.—Skull slightly thicker than normal, but of usual density. It is symmetrical. The dura mater is not adherent; the sinuses contain dark fluid blood and clots. The arachnoid is thickened, milky on the frontal and parietal lobes. The thickness and cloudiness of the arachnoid terminate abruptly at the line of the annectant gyri. There is no atheroma of the arteries of the base, which are of exceedingly small calibre. The pia mater is a little tough and thick, but seems to strip fairly. The convolutions of the frontal and parietal are slightly wasted.

Whole brain weighs 1,145 grammes, and is preserved in nitric acid.

Thorax.—Heart weighs 348 gms. No fluid in the pericardium. Valves competent, muscular substance firm. Left side contains fluid blood. Right side is contracted and empty.

Right lung, 1,015 gms. It is carnified. Over the pleural coverings and in the substance of the lung are numerous hard, tubercular granules, the largest of which is of the size of a small shot.

Left lung weighs 800 gms. It is adherent at the apex, and is also studded with tubercles.

Abdomen.—Liver weighs 1,821 gms., pale and soft. Spleen 183, normal. Kidneys: right, 153 gms.; left, 181 gms.

Pelvis and ureters in both are dilated, and full of bloody urine. Capsules strip freely.

Cortical substance pale, swollen, somewhat puckered.

CASE VI.—W. D. M., plumber. Admitted October 29, 1875.

Æt. 30. Married. Second attack. First attack in 1874. Duration of present attack, 4 weeks.

Supposed cause.—His father not leaving him his business.

Facts indicating insanity.—Expression vacant ; manner odd and childish. Fancies strange people are present in the room.

History.—Patient's second attack, and has lasted 4 weeks. He was in this Asylum during the first attack, and remained from July 4 to December 16, 1874. His wife says that after his discharge at her request he was for a long time out of work, and when he did get employed in his own trade as a plumber, though formerly a very competent workman, he was unable to do simple jobs. He would waste time, and finally leave his work incomplete. Subsequently he went to work as a labourer at some dye works, but, being peculiar, he was teased by his fellow-workmen and greatly annoyed. Four years ago he experienced great disappointment, because his father did not leave him his business, as had been expected. Since then he has become altered in manner, and the alteration has been more marked since his discharge. He has had paroxysms of excitement, but never struck anybody. On the other hand, he was always afraid of others striking him. His manner is odd and childish. He imagines that strange people are in the room, and that his wife is dead. He also points to policemen, and women in long ringlets, where none could be seen. Has shown no exalted ideas. Several years ago, after a slight difference with his wife, he threatened to drown himself. Has been sober. Has had 4 children, of whom one died of infantile convulsions.

Present Mental State.—On admission, patient was quiet ; slept well, but took his food badly. He has also wet the bed. Mentally, he is very stupid, and shows marked impairment of memory, both as to remote and recent events. He understands questions, but answers them irrelevantly, and in a mumbling, childish manner. He denies all the alleged delusions. When he speaks there is a decided tremor of the lips, especially under excitement. His tongue is tremulous.

General Appearance.—Hair brown ; pupils somewhat unequal, but active ; complexion swarthy ; expression bewildered. He is clean, and free from bruises. Locomotion is distinctly impaired. There is a red cicatrix on the outer aspect of the upper arm, and round the same are numerous spots of psoriasis guttata.

Respiratory and circulatory system, normal.

No trace of sores or cicatrises on the genital organs.

Diagnosis.—General paralysis.

Prognosis.—Bad.

Treatment.—Ext. Physostig. grs. $\frac{1}{3}$ ter in die.

November 15, 1875.—Very weak, demented, and dirty.

November 20.—Choked by a piece of meat getting into his larynx.

Died November 20, 1875, at 12.50 P.M. Examination 51 hours after death. Weather cold. Body fairly well nourished. Over the abdomen there is a layer of fat nearly $\frac{1}{2}$ an inch thick. The surfaces pale, and there are no bruises nor external marks. Rigor mortis is not present, and there is no hypostatic congestion.

On opening the larynx a piece of meat was found.

Head.—The skull is rather thick, and very hard. It is fairly symmetrical, and the bones composing it are pale. The dura mater is not adherent, but its sinuses are engorged with dark fluid blood. The whole brain weighs 1,489 grammes. There is opacity and whiteness of the arachnoid over the frontal and parietal lobes. The vessels of the pia mater are somewhat en-



Fig. 2



Fig. 1

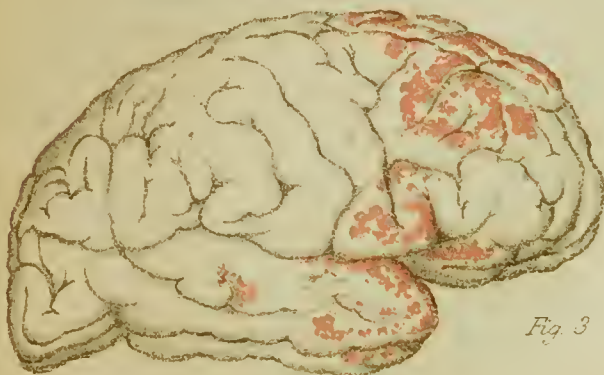


Fig. 3

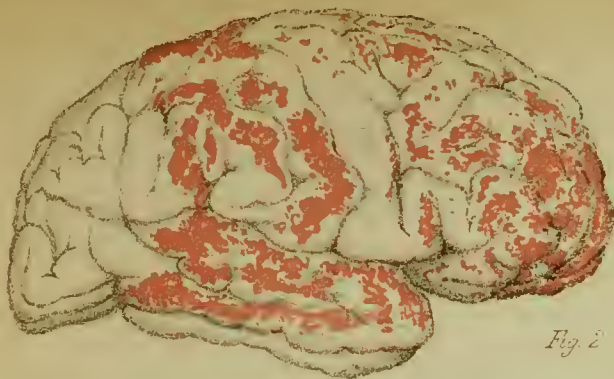


Fig. 2

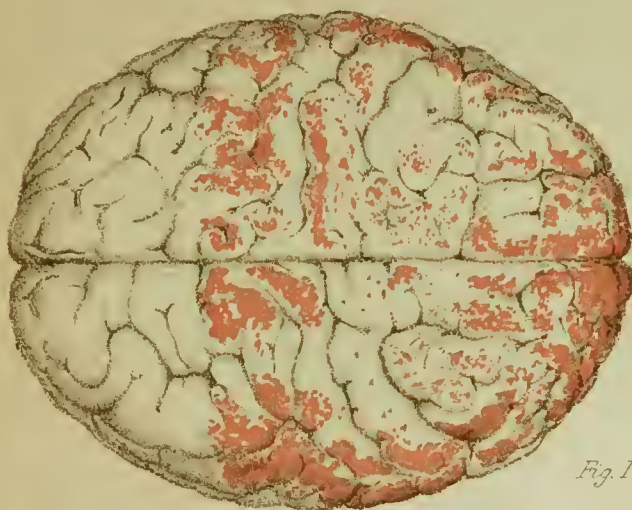


Fig. 1

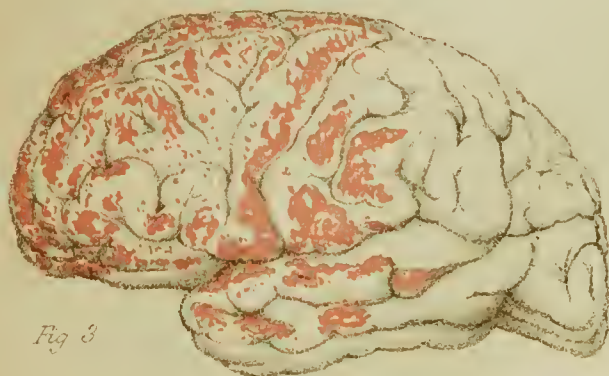


Fig. 3

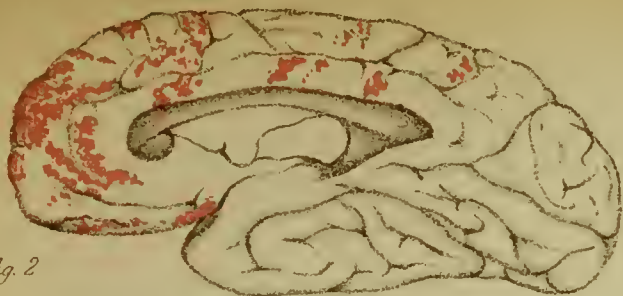


Fig. 2

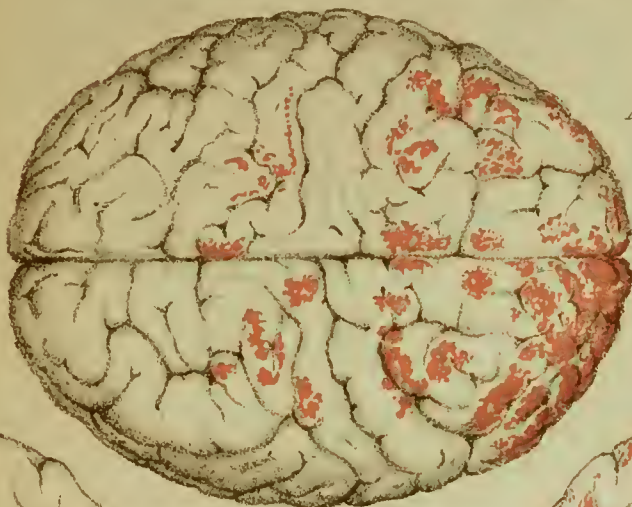


Fig. 1



Fig. 4



Fig. 5



Fig. 3



Fig. 1



Fig. 3



Fig. 4



Fig. 2

gorged, and the pia mater itself is thick, but seemingly strips freely. There is no atheroma of the large arteries.

Thorax.—No fluid in the pericardium. Heart weighs 290 gms. In the right side there is dark fluid blood. The left ventricle is contracted and empty. The valves are competent, but the inner surface of the aorta is rough and eroded by atheromatous deposit.

Right lung, 801 gms., free. It is dark-coloured and congested, but crepitant throughout. No tubercle.

Left lung, 784 gms., free, in the same condition as the right.

Abdomen.—Liver, 1,675 gms., dark-coloured, and contains a large amount of fluid blood.

Spleen, 185 gms., also dark-coloured and congested.

Kidneys.—Right, 162 gms.; left, 168 gms.

Capsules quite free. Both kidneys are congested, and the pyramids most so.

The brain is preserved in nitric acid.

EXPLANATION OF PLATES.

Plate III.—Fig. 1. Left temporo-sphenoidal lobe of general paralytic, showing aspect presented by areas of adhesion after removal of the pia mater.

Fig. 2. Upper aspect of cerebrum.

Fig. 3. Lateral view right hemisphere. Case of J. T., No. I.

Plate IV.—Figs. 1, 2, 3. Upper and lateral views of right and left hemispheres. Case of W. R., No. II.

Plate V.—Fig. 1. Upper aspect of cerebrum.

Figs. 2 and 3. Inner aspects of right and left hemispheres.

Figs. 4 and 5. Right and left orbital lobules. Case of F. S., No. III.

Plate VI.—Fig. 1. Upper aspect of cerebrum.

Fig. 2. Under aspect of whole brain.

Figs. 3 and 4. Right and left temporo-sphenoidal lobes. Case of B. W., No. IV.

Plate VII.—Fig. 1. Upper aspect of cerebrum.

Fig. 2. Under aspect of whole brain. Case of G. S., No. V.

Plate VIII.—Fig. 1. Under aspect of whole brain.

Fig. 2. Upper aspect of cerebrum. Case of W. D. M., No. VI.

As has been before said, these cases do not—in consequence of the very imperfect manner in which the reports have been kept—afford us any opportunity of tracing out a correspondence between the symptoms during life and the lesions seen after death. It would be useless to attempt

any analysis of them, and instead of doing so I think it better to indicate generally the correspondence which exists between the localisation of the adhesions and the psychical and motor symptoms in general paralysis.

In the first place, then, to what extent can we localise adhesions, as landmarks of the essential lesions? To what extent can we trace out their distribution over the surface of the hemispheres, define regions for which they have special affinities, or draw boundaries that they never transgress? To some extent we can already do this, and I shall proceed to give the results of my observations as to their topography. If, in doing this, I should make some statements that may not appear to be warranted by the illustrations here given, it must be considered that I am generalising from a much wider induction than these illustrations afford. Besides them I have had before me drawings of the brains of six other general paralytics, and full verbal descriptions of the brains of forty more. All of these I have kept in sight in preparing the very general scheme of the localisation of the adhesions here submitted. That that scheme will be subject to much modification and correction hereafter I am fully aware.

On taking a general survey of the stripped or prepared brain of a general paralytic, in which the adhesions are present, we are immediately struck by the fact, already mentioned, that they are confined to the anterior three-fourths of the brain, and are not to be seen on the posterior fourth. In all the six brains here delineated, the exemption of the occipital lobes from any trace of adhesion of the pia mater is very apparent. And this exemption is, as it were, only part of a wider immunity from visible pathological change which these lobes enjoy. In all varieties of chronic insanity, in which there is opacity of the arachnoid and thickening of the pia mater, that opacity and thickening terminate abruptly at the parieto-occipital sulci. Wherever there is wasting of the convolutions, that wasting ends with the annectant gyri and does not invade the three tiers of occipital gyri. And wherever there are morbid changes in the cortical layers revealed by the micro-

scope, these changes, whether in cells, fibres, vessels, or neuroglia, are less marked in the occipital lobes than in any other region of the cerebrum. These lobes, in short, in the insane, present morbid appearances of any kind less frequently, and to a less degree, than any of the other cerebral lobes. Their freedom from those special morbid appearances which we have described as characteristic of general paralysis is complete and invariable. I have never found genuine adhesion of the pia mater to the cortex behind the parieto-occipital fissure; and, as a rule, these adhesions are not found on the annectant gyri. It is not only the external surface of the occipital lobe that is exempt from adhesions; its internal aspect forming the cuneus or occipital lobule, and the posterior extremity of the uncinatus gyrus, are equally free from them. Over all aspects of the lobe, indeed, the pia mater remains thin, and may be removed, with care, without tearing away any of the grey matter along with it. I say, with care, because the very tenuity of the membrane in this region renders it easy to make abrasions, if scrupulous nicety is not observed in peeling it off.

But our general survey of the brain in general paralysis reveals to us another region, that may be said to be free from adhesions, and that is the island of Reil or middle lobe. Here, as a rule, the pia mater strips off most readily, and leaves the gyri operi with a smooth and unbroken surface. Very rarely have I noted adhesions in the island of Reil; and then there existed an advanced state of decay of the brain and other sources of fallacy. Ordinarily when the tough strong membrane spanning the Sylvian fissure is torn through, the pia mater may be removed from the insula with the utmost facility.

Turning now from those portions of the brain in which adhesions are not encountered to those in which they do occur, we may attend first to the frontal lobe which is undoubtedly their favourite site.

Here, if anywhere, are adhesions likely to be found. Here are they usually most numerous, and most firm, so that when they are torn through a considerable depth of

grey matter is removed, and a proportionally deep abrasion with steep rough edges is left behind. There is no subdivision of this lobe, and I include in it, in accordance with Turner's arrangement, the ascending frontal gyrus, in which adhesions do not occur. Over its superior external and internal aspects, and over the orbital lobule, they abound. In the marginal gyrus, where it forms the border of the great median fissure, both on its outer and inner aspect, they are generally very numerous, most so perhaps at its anterior extremity, where it bends downwards, and is recurved towards the anterior perforated spot. In the frontal gyri, properly so called, superior, middle, and inferior, they are also numerous and extensive; indeed, in my experience the anterior extremities of these gyri, forming the tips of the frontal lobes, constitute the areas where adhesions in cases of general paralysis are most invariably found and are of most marked character. In this locality the pia mater is often attached to the grey substance, not in scattered parts and patches, but over the whole of the summits of the convolutions, and tears away when removed, what is apparently, a half of the whole depth of that substance. In a few brains adhesions have not been found in this region, but in others they have been detected here when they could not be discovered elsewhere, and I am now more confident of being able to demonstrate them here than in any other region. On tracing the three parallel frontal gyri backwards, in cases where adhesions exist, these for the most part diminish in number until quite the posterior ends of the gyri, where they impinge upon the ascending frontal gyrus, is reached, when the adhesions again become decided, and partake of that marked character which is generally noticed in them in the ascending frontal gyrus. Dividing the frontal lobes on its superior and outer surface into three nearly equal sections by supposed lines running downwards from the median fissure, I should say that the adhesions in general paralysis are most pronounced in the anterior belt, that they are less frequent and severe in the second belt, and are again more pronounced in the third or posterior belt, which lies immediately in front of the fissure of

Rolando. At the posterior end of the inferior frontal gyrus, contiguous to the Sylvian fissure, adhesions are almost invariably found.

In the majority of cases adhesions are found over the slightly concave surface of the orbital lobule, where, however, the thickening and cloudiness of the membranes are never so decided as on the upper aspect of the frontal lobe. The adhesions in this locality are often more diffused, and less broken up and scattered than elsewhere. Their most constant situation is along the edges of the olfactory sulcus, and when they are torn through here it is not at all unusual for the olfactory bulb and peduncle to be torn away from their connections. The three gyri which enter into the formation of the lobule are all liable to adhesions, but the posterior one, which is adjacent to the central lobe, suffers from them less frequently than the external and internal convolution.

In the parietal lobes adhesions of an easily recognisable kind are generally found affecting all its four gyri; sometimes the adhesions are small and isolated, at other times they are large and closely grouped. Sometimes a single gyrus, such as the ascending parietal or supra-marginal lobule, seems to have escaped them altogether, and sometimes they are confined to one gyrus, such as the postero-parietal lobule. In one set of cases the adhesions are more numerous in the anterior half of the lobe; in another set they are more numerous in its posterior half. Occasionally they manifest a tendency to congregate in a line with the median fissure, and again they are massed near the horizontal limb of the Sylvian fissure. But whatever their distribution may be, the angular gyrus is but rarely free from them. We are not yet in a position to speak more explicitly of the arrangement or order of production of the adhesions in the parietal lobes, or to say more than that they occur there oftener than in any other districts of the cerebrum, except the frontal lobes.

In the temporo-sphenoidal lobes adhesions occur very irregularly, being sometimes found spread over the three series of convolutions, as in the cases of B. W., Plate VI.,

and W. R., Plate IV., and being sometimes limited to a few spots; as in the case of W. D. M., Plate VIII. In some cases the pia mater is not adherent to the cortex in any part of these lobes, as in the case of F. S., Plate V. The general rule seems to be, however, that there are two localities in these lobes which the adhesions especially affect, one being the anterior lip, and the other the upper margin, where the superior convolutions form the lower lip of the Sylvian fissure, and lie in contact with the inferior frontal convolutions. The thinness of the pia mater over the tempora-sphenoidal lobe, even after it has undergone pathological change, makes it very difficult to determine the existence of adhesions in this region, unless by the nitric acid process.

On the inner surface of the hemispheres adhesions are frequently seen anterior to the internal parieto-occipital, and to the collateral fissures. As has been already pointed out, they are copiously developed on the marginal gyrus, especially in front. They occasionally exist also on the gyrus fornicatus, the quadrilateral lobule, and on the anterior half of the uncinate gyrus. Not seldom, however, the uncinate gyrus is free from adhesions, and I have never seen any on the tentorial face of the hemispheres.

Both hemispheres are affected by adhesions to nearly an equal extent, and there is generally a considerable degree of symmetry in the distributions of the adhesions over them. On the whole they seem to be more numerous over the right than over the left hemisphere.

Having thus arrived at some general ideas as to the localisation of the adhesions in general paralysis, the next step is to sum up the symptoms of that disease, and to ascertain how far these corresponded with the results of experimental interrogation of the regions where the adhesions are found. This can only be done in the most brief and general way. Any attempt to unravel the complex phenomena of general paralysis, and to connect these with their anatomical substrata, to show how the symptoms are compounded, and to what sensory and motor centres their ultimate elements may be referred, would beguile us

into a disquisition of great length, for which our knowledge is not yet ripe. Here nothing but a faint outline of the more prominent symptoms is offered.

The symptoms of general paralysis are psychological and motor, and of these the psychological appear first in order of time. Soon after the psychological symptoms have declared themselves, the motor symptoms appear, and subsequently they advance together, though often at very different rates and in very varied combinations. In enumerating the symptoms, psychological and motor, I shall at present keep in view only the most typical cases of the disease, of which there are, of course, several recognised varieties.

Psychical Symptoms.—1. General restlessness and unsteadiness of mind, with impairment of attention; alternating with apathy and drowsiness.

2. A change in disposition and temper, and a general loss of self-restraint; at first as regards trivial social observances, and then as regards general conduct.

3. Impairment of the reflective powers, so that there is no logical and systematic development of thought.

4. General exaltation of thought, with a profusion of remembered images and ideas, and numerous extravagant desires.

5. Failure of memory and forgetfulness; at first of words, and then of events.

6. Delirious conceptions, and the transformation of desires into beliefs, these being generally connected with personal greatness and power.

7. Hallucinations of the senses, in which remembered sense impressions are so vivid and intense as to spread to the periphery.

8. Maniacal restlessness and excitement, in which present impulses and feelings instantly pass over into action.

9. Increased mental weakness, with the incoherent and fragmentary repetition of the false ideas previously entertained.

10. Failure of the senses, with more marked impairment of memory.

11. Complete fatuity, passing into coma and death.

Motor Symptoms.—1. Persistent contraction of the occipito-frontalis muscle, and some dilatation of the pupils, causing the eyes to be widely opened and the forehead wrinkled, and giving an expression of surprised attention to the face.

2. Persistent contraction and frequent tremors of the zygomatic muscles, giving a pleased and benevolent expression to the countenance.

3. Slight general muscular restlessness and unsteadiness.

4. Impairment of the power of executing fine and detailed movements, so that manipulative skill is lost while movements *en masse* are still well performed.

5. Fibrillar tremors of the tongue, and some loss of control over its movements, so that it is protruded with difficulty; is rolled about when protruded, and is suddenly withdrawn.

6. Twitchings of the nostril and upper lip, with frequent tremors of the latter.

7. Impairment of articulation, which is thick, and wanting in distinctness.

8. An alteration in the voice, as well as thickness and hesitancy in speech.

9. Loss of control over the combined movements of the hand and wrist, so that the hand-writing greatly deteriorates.

10. Changes in the pupils, which are at first irregularly contracted, and then become irregularly dilated.

11. An alteration in gait, which becomes unsteady; the more complex movements of the thigh, leg, and foot, and the balancing of the pelvis on the hip joints, being performed with difficulty.

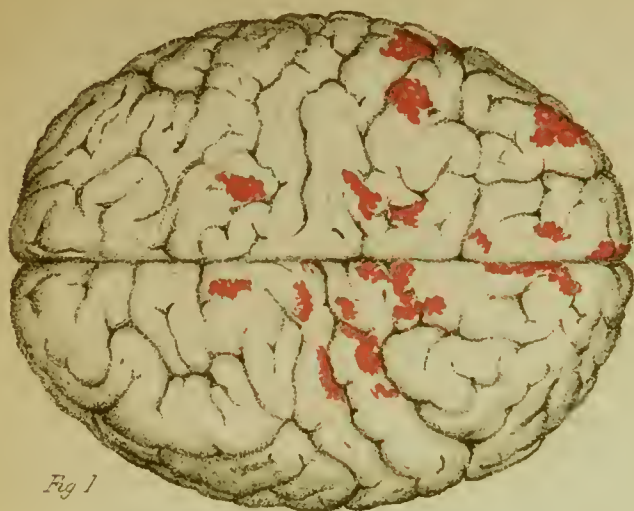
12. General muscular agitation and restlessness.

13. Gradual loss of power in the muscles of the face, tongue, neck, and limbs.

14. Spasmodic contraction of the masseter muscles, causing grinding of the teeth.

15. Convulsive seizures—most marked on one side of the body, and followed by transitory hemiplegia.

16. Loss of control over the sphincters.



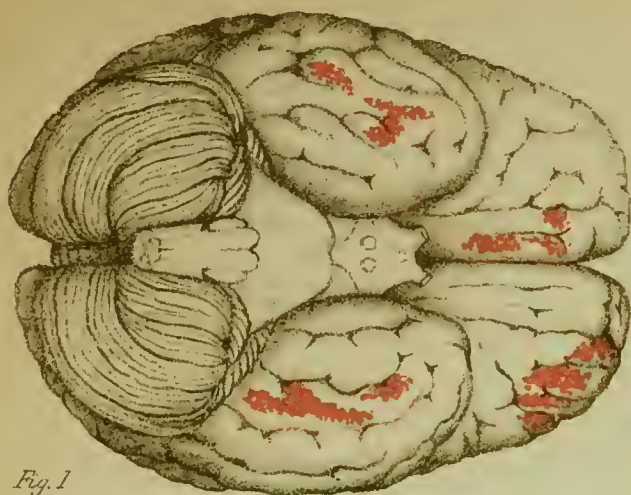


Fig. 1



Fig. 2



17. Complete prostration of muscular strength and helplessness, speechlessness, and difficult deglutition.

18. Contractions of the muscles of the limbs, and paralysis of the muscles of respiration.

The psychical symptoms unmistakably point to the primary involvement of the frontal lobes, which have been fixed upon by Ferrier as the centres of attention, and of the intellectual and reflective faculties, and where, as we have seen, adhesions are numerous and constant in general paralysis. 'In general paralysis,' says Westphal, 'the mental disturbance is from the first based on the foundation of mental weakness.' 'From the first,' says Salomon, 'there is a decided diminution of intelligence and of the power of judgment.' And the loss of inhibito-motor power, which diseased disablement of the frontal lobes would occasion, would produce a decided diminution of intelligence and judgment. The removal of these lobes in animals has indeed been shown to induce a state of mental degradation closely analogous to dementia. If the power of suppressing the actual movements into which an idea tends to diffuse itself externally be lost, there is no possibility of keeping up that internal tension in which attention consists, for external and internal diffusion are in the inverse ratio of each other, and that which is expended in motion is lost in consciousness. The necessary condition of attentive ideation being the arrest of outward movements, and the direction of the force thus arrested along the lines of internal organic cohesion, it follows that impairment of the power to suppress movements that have been initiated must involve the sacrifice of that high state of tension of the motor centres which is requisite to bring them into simultaneous action with the other ideational centres. Concentration of consciousness which demands complete inhibitory control becomes impossible. The power of directing thought is lost, and associative and sensory impressions are paramount in determining the succession of ideas. There is no deliberation, no abstract ideation, and new knowledge is not acquired in a precise and recoverable form. There is, in short, a general enfeeblement of the intellectual powers.

And this primary loss of inhibitory power explains many of the other symptoms of general paralysis, for it implies the loss of deliberation and the fruits of education and training, and the return of the mind to a more impulsive mode of action. Thus the alterations in disposition and temper so often seen at the outset of the disease are probably due to loss of inhibitory control. The patient is reduced to a more automatic sphere, has reverted to an infantile type, and his animal nature begins to assert itself. His instinctive appetites and desires are no longer held in check, but seek gratification, regardless of propriety, and in violation of those habits which education had conferred. Thus, at the outset of the malady, larceny is frequently committed, the patient at once appropriating whatever he covets, his action in doing so being conditioned by present feeling, irrespective of the associations formed by experience between such action and its consequences as future pains. And thus the sexual appetite, at some stage of the disease, manifests morbid activity. But its morbid manifestations here do not at all resemble those of satyriasis or nymphomania. They are not so violent, urgent, nor persistent. They do not arise as it were spontaneously, but are called forth by some affinitive impression. Their character suggests that they are to be ascribed to a loss of control over the sexual centres connected with the exercise of the generative functions, and not to direct pathological irritation of these centres. On examining these supposed centres—the occipito-temporal convolutions—after death, we find that they do not in general paralysis present any evidences, in adhesions, of having been subjected to direct pathological irritation.

And so it is with the appetite for food, which is a strong appetite, and must assert itself strongly when restraint is withdrawn from it. The general paralytic is ravenous, but it is in sight of food, and he does not go about incessantly craving aliment, as some lunatics do, in whom there are no indications of intellectual impairment, but in whom the centres of this appetite in the occipital lobes are, in all probability, irritated. These centres in the general paralytic are never marked by adhesions, and participate but little in

degenerative changes, and it is worthy of notice that the general paralytic does not refuse his food, but up to the last takes it freely.

The effects of the lesions of the frontal lobe, which I believe to be the earliest in general paralysis, are not at any time to be isolated from co-ordinate disturbances in other parts of the brain. The pathological change is gradually spreading backwards over the cerebrum, if, indeed, it does not originate simultaneously at several points on its surface, and the frontal lobes are in intimate functional connection with the motor regions. It is, probably when the posterior frontal and parietal regions are reached and implicated in the morbid process, that those delusive ideas of wealth and grandeur which are so characteristic of general paralysis are developed. The morbid stimulation of the motor centres, now more or less exempt from inhibitory control, must result in a flow of motor ideas, and probably in a sense of exuberant power. There is always pleasure in free muscular exercise, and this mounts into a feeling of delight when with that exercise there is a conviction of superior might and energy. In the general paralytic the higher motor centres are morbidly excited. He feels conscious of a sudden access of nervous power and is unassailed by fatigue, and the emotion of power which thus takes possession of him, and which is accompanied by outbursts of laughter, soon assumes the specific form of delusions connected with his own authority, wealth, rank, ascendancy, or accomplishments.

The order in which the motor symptoms of general paralysis present themselves, corresponds pretty closely with the order in which, according to Ferrier, the motor centres are arranged in the cerebrum from before backwards. Of course there are great varieties in the order in which these motor symptoms appear, and in the manner in which they are combined. In some cases one or more of them are never seen; in other cases they succeed each other most irregularly; but, as a rule, I believe they come in the order indicated, and are manifested through the muscles of the face, tongue, larynx, hand, arm, foot, and leg in succession. The centres for the muscles of these parts are all placed in

the posterior third of the frontal and in the parietal lobe,—in a region where adhesions are always abundant in general paralysis. Each motor centre affected by the morbid processes of general paralysis would at first, during the hyperæmic and inflammatory stage, be irritated and stimulated to a profuse reproduction of motor ideas, and to incontinent discharges, and would, finally, during the degenerative stage, be broken down, and have its functional activity abolished. And thus it is that we have at first in this disease, which is called general paralysis, motor symptoms which much more resemble chorea than paralysis. There are always tremors or clonic spasms of muscles before there is loss of power. The nervous discharges in general paralysis may be looked upon as transitional and intermediate between the deliberate, measured, and sustained discharges of health and the abrupt, profuse, and short-lived discharges of convulsive disease. Beginning in scarcely perceptible departures from the former, they end in becoming identical with the latter; for when a large number of motor centres, over a wide tract of cerebrum are involved in the morbid process in general paralysis, we have distinct epileptiform seizures, followed by hemiplegia, due clearly to exhaustion of the centres that have been so powerfully discharged, and in all respects resembling ordinary hemiplegia due to clot, except that it is transitory. Throughout the greater part of the course of the disease, however, the nervous discharges are something *sui generis*, and are displayed ordinarily in movements that are very different from those of voluntary or convulsive action. The movements developed by the pathological excitement of the cerebral centres are disordered in character, feeble in degree, and intermittent. They represent not sudden gushes, but frequent dribbling away of nervous energy, and they are greatly increased by every voluntary effort.

The incessant dribbling of nervous energy from the motor centres results every now and then in their exhaustion, and temporary partial loss of power in the muscles which are usually in a state of tremor and this loss of power from exhaustion, merges gradually in the permanent loss of power that is the consequence of the destruction of the cerebral

centre. The centres become enfeebled and isolated, so that they cannot act in concert with other centres, and then any strong impression made upon them induces not definite movements of desire or avoidance, but vague, feeble, and diffused muscular agitation. In the last stage of the disease there is universal powerlessness. The paralysis which followed irregular and disordered motor activity, and which invaded first those muscles most engaged in voluntary movements, reaches at last to those which are most automatic in action, so that even the respiratory and circulatory movements are endangered.

The sensory centres seem on the whole to be more frequently exempt than other parts of the cerebrum from morbid change. There is one of them, however, the angular gyrus or *pli courbe*—the sight centre according to Ferrier—on which adhesions are often seen. Now hallucinations of vision are the most frequent of all hallucinations in general paralysis; indeed hallucinations of any other kind, except these and others which may be shown to be connected with the muscular sense, are exceedingly rare.

It may be interesting to endeavour to indicate the effects of the morbid process of general paralysis acting upon the angular gyrus. Let us suppose that the inflammatory action which has originated in some other part has spread to this convolution. The centres contained in it will in the first place be stimulated, and there will be excitation of subjective visual impressions, probably accompanied by some of those reflex actions which are seen to result from its electric stimulation, such as restless movements of the eyeballs, which were noted by Dr. Clifford Allbutt in a large majority of the general paralytics examined by him, and contraction of the pupils, which constitutes one of the most familiar signs of the disease in its earlier stages. Past visual impressions will be recalled in a vivid manner, and these will probably harmonise with the pre-existing complexion of thought. If that has been bright and cheerful, then the visual reminiscences will be gay and delightful; if it has been dark and sombre, they will be gloomy and depressing. It is not, of course, meant that the visual reminiscences are

recalled in the angular gyrus, for it is obvious that, if this be the sight centre, here only the physiological representatives of optical characters, that is to say, light vibrations, are registered and retained, and that by these alone no object can be recognised. Most objects are known through several senses, and perhaps, also, through muscular adjustments; and every phase of the ideal persistence of an object must be stirred before it can be remembered. Several sensory and motor centres must therefore be called into associated activity before the idea of an object can be correctly revived, or each centre must retain and be able to recover numerous subsidiary impressions of a character different from those of which it is the chief focus. As the latter hypothesis only shifts the difficulty, we are constrained to adopt the former and to hold that in all but the simplest reminiscences several distinct centres are brought into combined action. No idea has, truly speaking, a single point of origin or revival in the brain, but every idea has roots extending into what are perhaps remote regions, and is dependent for its revivability upon every root that is struck at the moment of its acquisition. But in every idea, the roots convey to one centre or main root the primary quality of the idea, the point from which revivication originates, and in the case of visual ideas that main root is the angular gyrus. When, then, the angular gyrus is stimulated, visual ideas are revived, and as the hyperæmia and functional excitation of the centre increase, the revival becomes more and more vivid, the molecular thrill becomes more and more intense, and a faint reminiscence becomes a clear picture in the mind's eye. And then the molecular vibration which is the basis of memory overflows its ordinary basin and channels, and is propagated along the optic nerve to the retina, and there it re-induces a condition in some degree like the original impression which that vibration represents, and then we have insane visions or hallucinations which are outward reflections of the commotions within. But the vibrations in the angular gyrus which result in hallucinations are called forth by morbid irritation, and not by sensory impressions or associations, and they are therefore incongruous and out

of harmony. The darkness sparkles with diamonds, the walls are papered with bank-notes, the ground is paved with sovereigns, or ghosts start up, coffins are spread around, or terrible wild animals threaten to assail. And these revived sensations are accompanied by feelings of pleasure or pain, and gradually induce exhaustion of the centres in which they are revived. This exhaustion is followed by degeneration of the centres, which implies a loss of visual perception and defect of recognition. The patient gazes vacantly about him; if the finger is approached suddenly to the eye, the eyelids are closed; if an object is held up before him he can with some difficulty grasp it, but the object is not truly perceived, for if it be a watch he may convey it to his mouth and try to gnaw it with his teeth—if it be a book he may place it on his head. Later still, there is in most cases some impairment of vision, and in a few instances, those doubtless in which both angular gyri have been involved in the morbid process, complete blindness.

A CASE OF EPILEPSY

(UNDER THE CARE OF DR. CRICHTON-BROWNE),

REPORTED BY THE

MEDICAL OFFICERS OF THE WEST RIDING ASYLUM.

E. C., domestic servant, chargeable to Halifax, admitted on September 26, 1874.

Facts observed by medical man.—She is very excited, rambles on various subjects, says ‘that she would give me five pounds to cut her head off,’ ‘that she would kill her self,’ ‘that she wants to kill some one.’

Other facts communicated.—The surgeon to the Halifax Infirmary reports that she was very violent during the few days she was under treatment there.

Age.—28. Single.

Religious persuasion.—Church of England.

Second attack.

Age on first attack, 21. Has been in Northampton Asylum.

Insane.—Eight days.

Supposed cause.—Unknown. Is epileptic. Is suicidal. Is dangerous to others.

History.—The patient’s sister states that this is her second attack and commenced eight days ago. The first is said to have occurred when she was twenty-one years of age. She was then sent to the Northampton Asylum, where she remained for six years, and was discharged recovered. Before the first attack she is said to have suffered from ‘brain fever’ but made a good recovery and was subsequently in service.

While in service she had a bout of epileptic fits which issued in the attack for which she was sent to the Northampton Asylum. Two years ago she was discharged, and has continued well till about eight days ago, when during a bout of fits she was taken to the Halifax Infirmary, and thence to the Workhouse. In the Workhouse she has been very violent, attacking the inmates and tearing her own hair. She has refused food under the influence of a delusion. The fits are said to be epileptic. She has asked people to kill her, and has said that she would cut her own throat.

Family History.—Two sisters have been mentally diseased. An insane cousin on the mother's side died in the Northampton Asylum. Her mother died of consumption. The patient has been sober and steady. She has not had any blow on the head.

On admission.—She was first quiet and depressed, and then violent. She has had no fits since, but has threatened to kill people.

Present mental condition.—She is very confused, has evident difficulty in collecting her ideas, and to most questions answers 'I don't know.' She mistakes the identity of people around her, and can give no account of the origin, nature, or occurrence of her fits, except that she began to suffer from them some time after she had brain fever. She is incorrect as to dates, etc. Is a good deal excited and restless, and beats rhythmically with her hands.

Physical condition.—Hair brown. Eyes grey. Pupils equal and active. Conjunctivæ much congested. There is slight fixity of the features and stolidity of expression. She is well nourished.

Alimentary system.—Tongue clean, not bitten; appetite capricious; state of bowels not ascertained.

Respiratory system.—Normal.

Circulatory system.—Heart normal; pulse 108.

Genito-Urinary system.—Says she is regular; urine normal.

Diagnosis.—Epilepsy with homicidal and suicidal impulses.

Treatment.—Pot. Brom. ʒss., Liq. Ext. Ergot, ʒii. ter in die.

October 5, 1874.—During the night between Friday and Saturday last (three nights ago) the patient had a series of fits, and was found by the night nurse busy destroying her bed-clothes. During the course of Saturday she had six groups of three fits each. Two of the bouts of three fits were in succession, separated only by an attack of active destructiveness, during which she was unconscious. During the night she required to be placed in the padded room. During the morning of Sunday she had twenty-one distinct fits occurring in bouts of three each. After dinner she had three single fits, followed by destructiveness, and afterwards she lay in bed in a heavy drowsy state. About five o'clock grs. 20 of hydrate of chloral were administered, and she had some brandy. She then got up to tea, and from six o'clock to seven she had other three fits, after which she required to be held, to prevent damage from her violence. On Sunday night she was again placed in the padded room. As her dress was torn in the morning, it is presumed that she had had a series of fits followed by the usual paroxysm of violence and destructiveness. This morning she has had two bouts of three fits each before eleven o'clock, and during medical examination at that hour she dropped suddenly and without warning from her chair, and was convulsed. First the lips twitched violently. She foamed at the mouth, but did not bite her tongue. In about half a minute, the arms were thrown into clonic spasm. The convulsions affected both arms equally. The spasms affected principally the muscles of the upper arm. The humerus was struck repeatedly and forcibly against the floor. The legs were only slightly convulsed, and were almost immediately thrown into a condition of tonic spasm, the feet being held about ten inches above the floor. After about a minute of convulsive movements of the face and upper extremities, she inclined to roll over on her face towards the left. At this stage the right side of her face was more convulsed than the left. She now passed into a condition of tonic spasms. The body was rigid and the arms outstretched at right angles to the trunk; the hands firmly clenched and the

limbs immovable at the joints. The hands were held at about a foot from the floor when she was lying on her back. This condition lasted for about a minute. Her whole body became relaxed, and for another minute she lay in a drowsy unconscious state. Then a new fit commenced, presenting exactly the same phenomena, but shorter in duration. After a similar interval a third fit began, manifesting the same characters, but shorter than the second. After the third fit, when the muscles had been relaxed for about a minute, she gave a sharp chuckle, and commenced to make clutches at the carpet. As she could not get a sufficient hold she desisted, and seized her apron which she tore to shreds. She then sprang up suddenly, darted towards the window, and attempted to smash the glass. When restrained she struggled violently, and made repeated darts towards the windows on either side of the room. She was quite unconscious. The struggle lasted for about three minutes. She endeavoured to bite the hands of those who held her; her face was much congested and puffed; she ground her teeth slightly. After this she lay for about three minutes in a heavy semi-conscious state. At the end of that time she answered questions correctly. She said that she could not imagine what was wrong, asked if there had been a fight, and cried out that she could not tell where she was. When asked if she remembered anything about the commencement of the fits, she showed no recollection of anything relating to them, and a complete failure of memory regarding things which had occurred long previous to the commencement of the attack under observation. She said, as she had said before immediately after a seizure, that there was a clock in her head, and that she could hear it being wound up, and feel it excessively tight when the winding-up was completed. At the same time she says she hears a man's voice telling her that she must kill somebody, and subsequently kill herself. The voice is not that of any particular man, but it demands that she should kill somebody whom she is much attached to. She also hears bells ringing, and complains of fornication and muscular exhaustion, which are also very disagreeably present in the contents of the orbit. She asks

to have her hair cut off, and says that if it were removed the tightness would be obviated. (Her last act before regaining complete consciousness has always been to pull her hair out.) When asked to describe the position of the clock she defines the circle in which it is wound up, making a sweep of about four inches in diameter round the vertex, defining a horizontal plane. She believes there would be no culpability in murdering anybody at the command of the voice as long as it was somebody that she loved. She states that she once told the cook in her last situation—a woman of whom she was very fond—that she would be obliged to murder her sooner or later. She continually reverts to the racket of the clock. She is completely unaware of having had fits, and when asked if she has ever had such a thing answers ‘No’ in an astonished and somewhat indignant manner. In about ten minutes after regaining consciousness she was clear-headed, and even sharp and cheerful. In conversation she ascribed her first illness to the death of the man to whom she was engaged, and was emotional during the time of making the statement. She says that frequently everything becomes dark round about her, but she is unable to give any support to the notion that this may occur at the commencement of the fits. She says that for the last six weeks she has had the same feeling of muscular exhaustion and formication, and pins and needles in the eyeballs, which she experiences now after the bout of fits, and that ‘she could never understand it.’ The sensation in the eyeballs has, she says, caused her to lose much sleep. The nurse states that the patient never refers to the clock in the head or the incentives to homicide except immediately after a fit, and says that the former bouts were identical in character and progress with that now described.

October 7.—Her fits continue to be very numerous and severe. She is taking 45 grains of bromide of potassium thrice daily. Last night she had 5ss. of chloral. So far as can be known, she had one bout of fits during the night.

October 13.—At 10.20 P.M. yesterday the skin was very hot, and bathed with perspiration. Pulse 150. At

11.15 she had a bath, temperature commencing at 85°, and cooled down to 70°. Having remained in it 20 minutes she shivered, and was taken out. At midnight the temperature was 104·80°. Respirations 28. Pulse 128. Previous to the bath the temperature of the body was not taken; but subsequent to the bath it was palpably diminished from what it had been. At 12.20 A.M. to-day there was marked nystagmus, and great dilatation of the pupils on the application of a strong light.

October 14.—Temperature 104°. Pulse 120. The semi-comatose condition is not so deep.

October 18.—Yesterday the patient had recovered consciousness completely, and sat up; apparently there is no remaining impairment of motor power.

October 20.—She had no sleep last night. The fits keep off. She is rational, but the nurse has noticed the same restlessness which she manifested previously to the last bouts of fits. She sleeps very little. Yesterday she was ordered liq. bismuthi ʒ 1 thrice daily, as the tongue was very much furred.

October 26.—The night before last she had one fit. She had complained on the previous evening of the feeling of a clock in the head, as before. The periodicity is still observed. Yesterday she had 27 fits. Nitrite of amyl was given three times during the afternoon, and twice at night.

October 20.—Since last report the fits have continued, averaging about 18 during the day. In all respects they have presented similar characters, and have been accompanied by similar mental symptoms, as on the first attack of fits, before fully recorded. It is to be observed, however, with regard to the fits, that besides occurring in groups of three, they are in other respects peculiar. The phenomena are as nearly as can be observed as follows:—

1. Rolling of the eyeballs to the right, and slight general twitchings of the arms and legs (bi-lateral).

2. Rigidity of all the muscles, hands clenched, legs perfectly rigid (lasting about a minute).

3. Strong general convulsions (bi-lateral), in which she

usually rolls towards her left side. This is followed by deep laboured breathing and stillness; the legs and arms, however, remaining stiff and rigid. This is again followed by convulsions in the same order as before, and not until the three convulsive seizures are over, do the legs drop from their rigid extended attitude, and it is then known that the seizures are over. This morning patient was found to be in a heavy stupid state, not answering to questions, and not to be roused. Condition at 4 P.M.: Semi-comatose, the eyeballs being sensitive; remains perfectly still, and cannot be roused; breathing quiet; pulse very irregular, and occasionally intermittent, about 100. Pupils dilated and in a constant state of alternate dilatation and contraction; face very pale; cannot swallow; fits continue, but are more feeble. Ordered to omit nitrite of amyl, to have beef-tea and brandy enemata. Patient is in a very critical state.

November 3.—In a measure, at least, some of patient's symptoms described in last report seem to have been hysterical. She continued in the state described during the night, and the following morning was fed by the œsophageal tube. At about 2 o'clock she recovered consciousness; but in attempting to speak uttered only inarticulate sounds. About two hours after she spoke as usual, and took food; the fits ceased at midnight on the 29th ultimo, and have not since recurred. At present patient, though free from hallucinations and sensations of the clock in the head, is very much depressed, and openly expresses her intense desire to commit suicide in atonement of the dreadful sins she believes she has committed. As in the previous instance following the fits, her tongue is very much loaded, and she has a feeling of nausea. She is taking sp. ammon. aromat. and calumba. She sleeps fairly well, and takes liquid food.

November 7.—The effects of the attack are passing off, and patient has resumed her former condition. She complains of constipation, and was ordered a black draught. Tongue is clean.

November 8.—Is up and dressed to-day, is very cheerful.

November 14.—Patient continued well until yesterday. She was noticed to be restless, and did not sleep last night. This morning homicidal tendencies have shown themselves. She is restless and anxious. Ordered 30 grains of bromide of potassium every four hours.

November 15.—Her condition continues unimproved, and she is evidently undergoing the crescendo movement, which culminates in an explosion of convulsions. Is pale and very restless, and anxious-looking. She is acutely suicidal; wishes that the blunt knife with which she is cutting her meat were sharp, and asks reporter for a carving-knife, as she must kill some one. Asks the medical officer to put her to death. At the evening visit she exhibited a tendency to violence; snatched up her cup, and wished she could get a weapon to kill somebody. She has been extremely restless and unhappy all day.

November 16.—She continues to get worse. Says she hears the clock in her head (this is one of the last forerunners of a series of seizures). Ordered to continue the bromide every four hours, and to have 30 grains of chloral hydrate at bedtime, as she remains sleepless, and it is to be hoped that the exhibition of chloral will avert the occurrence of the convulsive attacks.

November 18.—Patient had three convulsive seizures last night. She is menstruating this morning.

November 19.—The seizures continue both day and night, and recur at times, corresponding to the hours at which they occurred in her former attacks. She continues the chloral at night, and 30 grains were administered to her at the morning visit, as the occurrence of fits was looked for in about half an hour.

November 24.—From 19th to 23rd inst. she had 72 fits during the day. The clock-ticking has disappeared, and she has resumed her usual condition.

December 7.—On the 3rd inst. peculiarity of manner came on, then restlessness and excitement, with loss of sleep. At the same time she heard the 'voice' and became violent; no fits. From December 12th to 17th she had during the daytime 156 fits.

December 19.—Patient has just emerged from an attack of epilepsy following, with regard to duration and character of the fits and other features, almost exactly the course of those before reported. The attack has been followed by stupor, lasting in this instance a day and a night. Like the others, it has been coincident with the monthly period.

January 9, 1875.—Last Monday excitement came on again, and continues in a wild form. She has the sensation of pins and needles in the eyes; hears the voice, but not yet the clock.

January 15.—Patient is again going through the periodic attack of fits. In all essential points it is identical with those before fully described. It is now the 4th day of the attack which was, as usual, preceded, and has since been accompanied, by the feeling of a clock in the head; but this time the sensation came on only just before the first fit. Having been desired to inform the nurse when she felt the clock, she suddenly, and in the evening, when in bed, screamed out to the nurse to come to her, as the ‘clock had come;’ and almost immediately went off into a severe fit.

January 16.—Yesterday at 1 P.M. a powder of 25 grains of sulphate of zinc was administered, producing but slight vomiting. The fit, at about this time, came on ten minutes earlier than usual, but was slightly modified in its character. At 8 this morning 25 grains of Pulv. Ipecacuanha were given to her. Copious vomiting followed; a batch of three fits came on 8.40 (ten minutes earlier than on the 15th), and a second batch of three fits at 9.20 (an hour earlier than usual).

One P.M. Succus Conii 5 iv. given. Ten minutes afterwards the fits came. They presented the usual features, but were decidedly less powerful, the after ‘rush’ being very feeble. Ten P.M. patient is quiet and composed, and expresses herself as feeling better.

January 17.—No fits occurred during the night. This morning the patient is again quiet and composed. The ‘clock’ has left her, and the attack is over. It will be seen that it is the shortest that has yet occurred, and much the mildest

as regards constitutional disturbance and after-effects. Chloral (gr. xxx.) has been given each night during the attack.

January 20.—Yesterday the chloral was discontinued; bromide resumed. No more fits have occurred, but the patient complains a good deal of pains in the head, and has fainted on three or four occasions.

February 1.—The patient has not cleared up as usual. Since the attack she is irritable in temper, and complains of uneasiness in her head.

February 3.—Patient still continues very irritable, and on the slightest offence becomes violent, striking those about her. On Sunday she stated that she felt something turn over in her head. To-day she heard the voice. She is dull, heavy in manner, depressed in spirits, pale in colour, and very suicidal in inclination. Ordered Tr. Eucalypticus Globulus.

Yesterday morning the fits came on, and have recurred as usual.

February 10.—Had three fits this morning. There is stupor, muscular flaccidity, the pupils both dilated, right larger and fixed. During the medical visit she had two fits; both commenced with violent twitchings at the right angle of the mouth. The head was drawn slowly to the right side—clonic spasm—gradually becoming more and more violent; affecting both the right, and then the left arm and hand. The eyeballs were drawn under the upper lid. The feet were raised from the ground; the left first, and then the right, and both became rigid and quivered. The whole fit lasted about ten seconds, and terminated suddenly, with complete muscular flaccidity.

February 11.—Patient lies in bed in a semi-cataleptic condition, eyes fixed and staring, motionless, and refusing to take food. When raised and not supported, she falls to the right side, as if that side were weak. There is great irregularity of the pulse. Reflex action cannot be induced by tickling the soles. Another batch of fits came on during the medical visit; the right corner of the mouth was first convulsed, then the right arm, then the right leg, and then clonic spasms passed to the other side of the body.

Feb. 12.—Number of fits by day 107. Fit period ended.

Feb. 22.—Since the last attack patient's manner has been peculiar, and she has not cleared up as usual. She is also somewhat childish.

March 3.—Patient has been for two or three days in her usual quiet state. Excitement has now come on, with a sensation of pricking in the eyes associated with injection.

March 12.—Yesterday the attack ceased, having lasted the usual period. She had in the day-time 111 fits and no ecstasy. She took *Eucalyptus globulus*.

April 7.—A new bout commenced on the 2nd inst.: it lasted till the afternoon of the 6th. She had 129 fits during the day-time, mostly in threes, but eight times in sixes.

On April 6th she had discontinued the *Eucalyptus globulus*, which she had been taking for some time, and on April 25 was put upon 3 gr. doses of quinine, having during the interval been without medicine. On the 29th and 30th the fits occurred with great intensity. On the first of these days she had 27 (day) fits, and on the second 33. She again commenced to take Tinct. of *Eucalyptus globulus* on May 1st. On that day she had, between 9 A.M. and 6 P.M., 36 fits. The fits were much more severe than any she had previously suffered from, and the number was greater than on any day since her admission. Dyspnœa was most threatening, and it was considered necessary to take very active measures by artificial respiration to prevent a fatal issue. Up to this time she had had during the three days (under Quinine and *Eucalyptus globulus* treatment) 96 fits.

By the experience of former attacks it was certain that the fits would last for at least two and at most three days more. As the administration of chloral had not been on former occasions pushed to a full extent, it was resolved to administer it to such a degree as was compatible with safety. To begin with, 30 grains were given at 7 o'clock. At 7.35 she had 6 fits occurring in one bout; at 9 o'clock she had a similar series of 6; and at 9.45, 30 grains of chloral were given by the rectum. At 11 P.M. she again had 6 fits. There was very little difference in the nature of these fits from the description several times recorded, but they were

very intense *quoad* muscular action. During the excitement following upon the fits it was noticed that the paroxysms of impulsiveness were of the same number as the preceding fits. After six fits there were six attempts to spring from the bed on which she lay, and on the following day, when the attack consisted of only three fits, there were only three subsequent struggles.

On waking up, after the excitement was over, she always complained of severe lancinating pain in the region of the heart. The struggles following upon the fits were somewhat tetanic in their nature. The head was first thrown back, and the chin pointed. The cervical muscles were in a state of tonic spasm, and the back was arched so that a pillow could have been slipped under it; there was decided *opisthotonos*. This lasted for about thirty seconds. Then the head was thrown violently forwards, and brought instantly back again with a heavy thud upon the pillow. This movement was repeated five or six times. She then tossed her head and shoulders five or six times from side to side, and all the time struggled severely with all her limbs. Each new attack of excitement was preceded by a twitching of the right side of the face. (She has been observed, however, to have had on some occasions, twitching of the left side, the right being at rest.)

After outbursts of excitement, all of which resembled the one described, and followed each other after barely a minute's interval, she became calm. The muscles of the limbs and trunk were gradually relaxed. She breathed heavily, and forced a large quantity of foam from the mouth, woke up, screamed, and complained of severe pain in the region of the heart. Between the end of the last struggle and the return of consciousness there was a period of two minutes. On making enquiries it was found that *opisthotonos* had been observed in several previous fits, but never so severe.

May 2.—At 11, and again at 12.30, she had six fits followed by six outbursts of excitement. It was known by experience that unless the patient had been restrained during the excitement, she would have made a sudden and

desperate attempt to kill those around her by direct assault, by biting, or by strangulation. Up to 12.30 she had, since the chloral was administered, 24 fits. The quantity of the drug given was still regarded as insufficient, and as sickness was present and made the quantity retained uncertain, the administration was carried on by the rectum. Tinct. of catechu and brandy were injected along with the drug, the former not to control a certain amount of diarrhœa which had been present all day, but to ensure the retention of the drug. At 1.25 A.M. the patient had 45 grs. of chloral by the rectum; almost immediately she fell asleep, and slept from 1.30 till 2.35 A.M. At 2.45 she had an attack of six fits and six struggles. Between the beginning of this seizure, and the beginning of the preceding one, there was an interval of $2\frac{1}{4}$ hours, almost double the length of any interval which had occurred during the previous day. Not only were the convulsive movements less severe than before, but the whole time occupied by the fits and the struggles was less. In the preceding bout the time occupied by the whole process was 45 minutes; in this it was 25 minutes. It will be seen that the fits commenced in the latter case exactly an hour after the administration of 45 grs. of chloral. The patient was visited at 3.30, and 45 grs. more of chloral were ordered; it was given by the mouth and vomited; 3i was given by the rectum at 3.45 A.M. The patient fell asleep at 4 A.M., and slept quietly till 6 A.M. At that time she remained awake for ten minutes, and was composed and conscious. Then she fell asleep, and slept till 7 A.M.

That the cessation of fits was not the result of exhaustion or coincidence was proved by the fact that an accidental omission in the administration of chloral hydrate was followed by a return of the arrested fits, and that the resumption of the medicine produced a lull, which, on the medicine being again omitted, gave way to a new outburst. At 7 P.M. on May 2 she was ordered 45 grs. of chloral by the mouth, but vomited it at once. No provision was at that time made for giving more, and the consequence was that for three hours from the time that the effect of the 3.45 dose had passed off she had none of the medicine. At 9.25 A.M. she

had three fits, showing that the action of the medicine was perhaps still sufficient to reduce the number from six to three. At 10.45 ʒi dose of chloral was injected by the rectum, but unfortunately before any of it could enter the system she had six more fits in one bout. At 10.50 they were, however, much modified as to severity. At 11.55 she was under chloral again, but had a bout which was limited to three fits. At 1.30 she had had no more fits, and was ordered another drachm of the drug. At 4.5 P.M. she was quiet and composed, and had had no fits since 11.55. She was very drowsy, and complained of great muscular pain. At 5.40 she was somewhat restless, and her pulse was quick; she was ordered another drachm by injection. At 10 P.M. she was quiet, sleepy, breathing slowly, and with a pulse of 111:

She slept without waking till 2.40 A.M., when she had six fits and six struggles in the presence of the medical officer. They were very mild, and lasted twenty minutes. It was observed that before several seizures her pulse and her respiration got much quicker than when she was fully under chloral. 60 grs. were injected at 3.30 A.M. She slept from 4.40 to 7.30. At 8 A.M. she had 45 grs., and slept; at 10.30 she had six strong fits. It was observed, with relation to this last administration, that whereas ʒi doses of chloral kept off the seizures for 6 hours, 45 grains could not be depended upon for more than $2\frac{1}{2}$ hours. There being no contra-indication, it was resolved to administer ʒi doses every 5 hours if required, and if no bad symptoms appeared. During the whole day she was kept under chloral, one dose of ʒi having been given at 11.20 and another at 4.20. She had no fits, and breathed calmly and slept comfortably. Several times her pulse was counted at 132. At 10 P.M. (May 3) she was found sleeping comfortably, and as the period of her fits was expired, no more chloral was administered. Her pulse was 140, respirations quiet and low in proportion.

May 4.—On this day it was found that during the night she had slept almost continuously, had had no fits, and had taken milk freely. The pulse at 10 A.M. was 118, and

the respirations were calm and regular. At this time she was still under the influence of chloral, and the question had to be determined whether or not it would be necessary or safe to continue the administration of the drug. Taking the former bouts as a criterion of the number of the fits and the length of time over which the attack should extend, it was known that in all likelihood she had reached the end of the seizure. Still there were three occasions on which the bout had lasted six days, so that it would not be altogether exceptional if fits were to occur on this (the sixth) day of the present bout.

As she was fully under the influence of chloral, and as there was only a probability of the recurrence of a serious epileptic condition, it was considered advisable to discontinue the drug.

At this point another consideration arose. Had the chloral, instead of antagonising the epilepsy, simply deferred the occurrence of the fits? Would the fits which had been arrested run their course after the withdrawal of the medicine? The occurrence of six fits at 11.5 and of three at 1.25 on May 4, 19 and 21 hours respectively after the administration of the last 5i of chloral, gave some support to the latter view, but the reduction of the last bout to the number of three at such a long interval from the use of the drug, and the complete stoppage of the fits for that time, showed that these fits took place on the sixth day of the bout, because the seizure, instead of being a five-day one, resembled several preceding ones in being of six days' duration. There can be little doubt that a 5i dose of chloral given on the morning of the 4th would have warded off the nine fits which took place on that day. Still, as the seizures could not have been foreseen, and as in the event of their non-occurrence the chloral might have caused serious effects, it would have been rash to have administered it. When examined on the morning of May 4 she was found to have a very soft blowing murmur at the base. This was, no doubt, hæmic, and could have no relation to the anginous pains which she frequently complained of when she regained consciousness after a bout of fits.

Throughout the whole seizure the patient's pupils were unequally dilated, the right being more so than the left. Sometimes the right and sometimes the left side of the face was most twitched in convulsions. At four o'clock on May 4th, almost three hours after the occurrence of the last fit, she was quiet, drowsy, and complained of great muscular pain. At 10 P.M. the patient had still had no fits, but she was frequently starting on account of pains spoken of as resembling knife-stabs in the region of the heart, and accompanied by great oppression in the chest movements. She spent a fair night between the 4th and 5th. On the morning of the 5th her pulse was 140, at 12 noon 108, and from this time she continued to improve, but for several days the tongue remained very dirty, and somewhat dry. On comparing the record of the present with past bouts of fits, it will be seen that chloral exercised an important action in limiting the number of the seizures.

The following Table will bring out the comparison between the three days of the April and May seizure, during which the patient was chloralised, and the corresponding (last) three days of other attacks :

Date	Duration	Number of Fits in Last Three Days
October . . .	6 days	42
November . . .	6 "	42
December . . .	6 "	78
January . . .	5 "	75
February . . .	5 "	61
March . . .	5 "	81
April . . .	5 "	75
May . . .	6 "	27

It will be seen that during the administration of chloral the number of fits was less (during the last 3 days) than on any occasion, even shortly after admission, when the fits were much less numerous and less severe than they have latterly been. At the same time it must be remembered that a definite reason has already been assigned for the occurrence of 12 fits on May 2nd, and 9 fits on May 4th, so that the only fits that occurred, while the patient was in

reality being treated by chloral, were 6 upon May 3rd. One thing is particularly worthy of notice. The opisthotonos, which marked the commencement of each struggle during the period of excitement, was at once controlled by chloral, but was again present when, 19 hours after the administration of the last dose of the hydrate, she had 6 severe fits. Following upon this—April to May—there was no ecstatic condition.

May 11.—Got up yesterday; feels much better. Tongue cleaning. The pupils are now normal in diameter and equal. Bismuth and hydrocyanic acid, which had been ordered for sickness towards the end of the attack, discontinued.

May 26.—On the 23rd she became a little restless, peculiar, and flighty. This has gone on increasing, and she has spoken of committing suicide and homicide if she gets the chance. There are twitchings of the eyes and pain in them, and a voice is heard by her telling her to kill somebody. As yet there is no apparent injection of the eyeballs.

May 28.—An attempt was made to ward off the attack by chloral, but ineffectually, possibly owing to the fact that she was not brought fully under its influence. She had four doses before the onset of the fits, two given by mouth, and two by injection. The last was given yesterday afternoon at 4 P.M. (strength of dose, 45 grs.). It proved quite ineffectual in procuring sleep, and the fits came on at 7.50 P.M., preceded as usual by the 'clock.' She had 3 fits up to 8 P.M., and another at about 9 o'clock this morning of the usual character.

June 2.—Yesterday evening the attack was over, having lasted five days. With regard to the time of occurrence of the fits, considerable variations and irregularity have been manifested, owing to the employment of chloral injections. The drug, as before recorded, did not prevent this attack, but the fits having set in in every instance in which it was given, the approaching fit, and sometimes even two attacks, were prevented. Every separate attack could be thus warded off at pleasure. As during the attack, however,

her general condition was never alarming, no attempt was made to ward off the fits entirely, but only to a certain extent each day. With regard to the phenomena presented by the fits in this attack, they are shortly as follows:—

1. Restlessness, vacancy, peculiarity of manner, preceding the fit for about three minutes.

2. Onset of the fit: working of both eyes, twitchings of both angles of mouth, working of both arms and legs (the preliminary tonic spasm at present can hardly be observed).

3. Tonic spasm of the muscles on both sides of the body, with a scream.

4. Clonic spasm again.

5. Relaxation of the muscles (fit over). The fits occur either in threes or sixes. But, in any case, the close of the last fit is manifested *by closing of the eyes*, which, throughout the attack, had been kept open. She remains thus, as if in sleep, for about 3 or 4 minutes, when the following phenomena occur:—

1. Eyes open suddenly, staring fixedly into vacancy.

2. A frightful struggle (the patient all the while unconscious), lasting about $\frac{1}{4}$ or $\frac{1}{2}$ minute—and this repeated 3 or 6 times, in accordance with the number of the preceding fits. Eyes open and glaring all the time, until the last struggle is over, which is made known by *closure of the eyes* and sudden calmness. She remains, as if in sleep for about 6 or 8 minutes, and then suddenly wakes, is composed, and asks where she has been.

June 3.—To-day ordered potass. iodidi. grs. xx., thrice daily. It will be seen that the fits have now, to a great extent, lost their one-sided character.

June 16.—She awoke suddenly last night, apparently aroused by the voice saying ‘kill them, kill them,’ which was repeated over and over again for an hour or more, until she was so harassed that, tired and prostrate, she fell asleep. The voice, she says, is a deep bass voice, and appears to her to proceed from the room in which she lies. There are noticed as concomitant symptoms a tingling of the ears and a perceptible flushing of the face. She hears the ‘clock.’

June 17.—This morning she is in a state of great agita-

tion, and declares she is tormented by the voice, saying she must kill herself or others. The attack appears to be coming on in the usual manner.

June 28.—Yesterday evening the fits again came on, showing the usual features. Last night at 7.10, and shortly after a fit, the temperature had risen to 100° , and the thyroid gland was noticeably engorged, and a distinct depression was seen along the coronal suture, as if the bone were separated along this line. The fits occurred, as usual, through the night, and left her this morning in a terribly suicidal state.

June 29.—The attack is now over. Her temperature has been taken every morning since the 8th of June last, when it was slightly below the normal, viz. 98.2° . From this date there was a progressive elevation until the 19th, when the rise became very decided, and remained about one degree above normal until date of the first fit, viz. the 24th). On June 26 and 28 observations were taken with the thermometer repeatedly through the day, and the following phenomena appeared:—Immediately preceding the attack a rise of temperature occurred of 1° to 1.2 Fahr.; and at the termination of the struggle a still further rise, often to the extent of one degree, or even more. The temperature then slowly fell again to 98.6° , and on one occasion as low as 98.2° , except when two fits succeeded each other quickly. On the occurrence of six struggles at 3.50 P.M. the total elevation of temperature recorded was fully 2.2° , slowly falling to 99.20° during the following hour and a half. The previous administration of chloral invariably arrested the rise, usually occurring before the fit; but at the exact moment when the fit was expected, instead of any convulsive seizure, there was sudden profound sleep, and a rise of about six-tenths of a degree. On the 28th one dose of chloral only being administered, the temperature remained persistently high, but the rise and fall during the seizure and half an hour afterwards corresponded with the results obtained on the 26th. For some 10 or 15 minutes (except when chloral had been given) before each seizure, the advent of the attack was preceded by *restlessness*, marked rise

of temperature, and a suddenly quickened pulse. She is left very prostrate, the fits having now ceased.

July 3.—The temperature was taken to-day at 1.30 P.M. and at 6.30 P.M.—periods corresponding to the time when her fits occurred last week—on the first occasion the temperature was 99.2°, on the latter occasion 100.4°, at other periods of the day (not corresponding to the date of a fit) the temperature was 98.4°. It was also noted that, as these periods arrived, she became suddenly and greatly depressed, often starting up and complaining to the nurse of her low spirits.

August 2.—A second series of observations was commenced on the 13th ult. with pretty nearly the same results, viz. gradual rise of temperature towards the advent of attack, great irregularity and febrile commotion during latter 2 or 3 days. On the 22nd and 23rd—that is, the second and third days of her attack—observations were made every five minutes throughout the day to note any fallacy in the previous conclusions. It will be seen that the results entirely accord with those previously noted, viz. rise of temperature immediately preceding the attack; but in these two days the temperature throughout the day never fell quite to the normal point, remaining constantly elevated.

August 25.—She has passed through another bout of fits, in which she has been treated principally by hyoscyamine. The drug was at first administered in 2 gr. doses, 2 doses of that size having been given in one afternoon. The result was the reduction of the fits to 13, as compared with 30 on the previous and succeeding days. Yesterday, which was the last day on which the fits were likely to occur, she had $\frac{1}{2}$ gr. doses every two hours. This dose almost entirely warded off the fits, and induced a mild cheerful mania, associated with great muscular prostration. She said she felt as if she were ‘pinned down’ to the bed. She had 6 very feeble fits during the day. Under the influence of the hyoscyamine many modifications occurred in her symptoms. Several times she had an isolated fit, which condition had never occurred before. Sometimes she had bouts of excitement without fits, fits without excitement,

and on one occasion she rolled over on one side and gave a cry, as if in a convulsive attack. This she repeated three times, and immediately after had three of her usual fits.

September 14.—After the usual interval another attack of fits is manifesting its approach. The iodide of potassium has quite failed to arrest or modify them. To-day it was discontinued.

September 28.—She has got over the last attack remarkably well, with no stupor remaining. The iodide has been resumed, as it has been thought possible that it may have had some effect.

October 2.—Yesterday she was noticed to be flighty and peculiar, showing that another attack is brewing.

October 14.—Last night at 11.5 the fits came on. She had nine fits; the struggles were so feeble that she did not require to be held.

October 15.—Yesterday she had 18 fits; they all occurred three at a time, and the subsequent struggles were extremely slight. Last night, as well as the night before, she slept well in the interval of the fits; and last night she had 6 fits, the struggles being slight and easily controlled. There is a very marked improvement in this as well as the last attack, in the present instance manifested by the following indications:—She sleeps well in the intervals of the fits; the mental symptoms are much less severe; the struggles following the fits are very slight; no clock has been heard sounding, as formerly described, and the voice, if present, is only occasionally heard. No special treatment has been adopted this time, but she continues taking the iodide of potash. During this attack, unless occasion demands, it is decided to allow the fits to have full play. A fit has just been observed; its duration has been 5 minutes. The phenomena recorded on previous occasions can all be recognised, but in every instance they are extremely weak and modified.

October 18.—Two days ago the attack came to an end, and the fits ceased; so that this attack was of two days' less duration, and the fits have been milder than usual in every way. She is up again, and very slightly depressed. It is quite evident the attacks have become fairly broken, and

there seems to be a good prospect of their discontinuance entirely. The following facts lead to the belief that the attacks depend on the condition of the uterus. When first taken to Northampton Asylum she was not regular, but on being discharged well she was regular. When first she came here menstruation did not occur at all for the first two or three months, and was then very slight, then abnormally dark, and sometimes excessive, all this time the fits being regular and very severe. Of late again menstruation has become regular, and more natural in every way, and with this there has been a gradual modification of the fits, as recorded.

November 3.—She has tided over three days now, during which she has usually been peculiar, without any symptom of the on-coming of the attack, as previously described.

November 6.—There is not the slightest symptom of the attack, but menstruation is not due till next week. She has got thinner, and states she is always in better health when she is thin.

November 15.—She has gone over an entire period now without any appearance of fits or any change for the worse. She appears perfectly well mentally.

November 30.—She continues well; medicine (iodide of potassium) discontinued.

January 28, 1876.—Coincident with her menstrual period, she has had a bad attack of depression, not without cause, but greatly exaggerated, with menorrhagia. No fits have occurred, and with the passing off of the menstrual period she has resumed her usual condition. She has been again put on iodide of potash.

February 8.—She is quite well again; the iodide of potash mixture to be stopped.

March 1.—Discharged; recovered.

NOTES
ON THE
THERAPEUTICS OF SOME AFFECTIONS
OF THE NERVOUS SYSTEM.

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THE division of the diseases of the nervous system which will be considered in this paper is that of the asthenic forms—the maladies associated with imperfect nutrition. They form a large class of maladies very commonly met with amidst the sane, especially in the out-patient department of our hospitals and dispensaries. The rules which have been formulated from experience for the relief of such patients will apply, with some slight modifications, to a large division of asylum patients. The factors in action, it will be seen, are necessarily the same in each, especially in female patients. It is indeed in women that we find so commonly and so pronounced the maladies about to be discussed. It may be said as a broad rule that the diseases of women are *par excellence* diseases of imperfect nutrition as compared to the diseases found in men. A great deal of this is due to their sex directly; a lesser but no insignificant portion is attributable to their habits and mode of life. In man, too, asthenic and anæmic states are found, but to a much less degree, and the circumstances which furnish these conditions

are such as to lend further significance to the causal relationship of these states of impaired nutrition.

That the nervous system should suffer in various ways from conditions of general mal-nutrition is readily intelligible. It is the most highly vascular of all parts of the body, and the demands upon it are incessant and often excessive. As a consequence, we find either central affections or peripheral disturbances. We may have headache, frontal, vertical, or occipital; or neuralgia, very commonly facial or intercostal. There are, however, other disturbances of the nervous system produced than painful affections. Perhaps the psychical pain, the sense of misery, of depression, of low spirits, is more truly painful to the sufferer than the physical pain, of which, however, more ready complaint is made. This condition, so common in women who are imperfectly nourished, is not far removed from melancholia, and both are amenable to the same line of treatment. A typical patient of this class will be found to present the following combination of objective and subjective phenomena, a certain amount of variation being assumed as necessary according to the differences of the several patients, and the individual characteristics of various organisms. There is a certain pallor of the countenance, and a listlessness of gait, a certain amount of lethargy, indeed, and that, too, where the neural diathesis is well marked, and the system endowed with considerable energy. The first complaint is usually of pain somewhere, most commonly under the heart, and as there is not rarely some palpitation as well, the patient is usually impressed with the notion that some disease of the heart is present. The next complaint is very frequently of indigestion, of pain after eating, with a certain amount of irregularity of the bowels. The tongue is often clean, with a certain silvery fur, and the impressions of the teeth very distinct around the edges—a state of the tongue very frequently found along with menorrhagia. At other times there is a certain amount of fur along the mesial line, but the tongue is rarely loaded with it. When it is so loaded the liver is generally found to be readily disordered, and the patient is subject to the well-known bilious attacks. There

is vertical headache very frequently; at other times it is frontal or occipital. The patient is low-spirited, miserable, despondent, and greatly given to crying. If asked, 'Do you feel ready to cry?' a flow of tears is at once evoked. There is great emotional susceptibility, and morbid psychical hyperæsthesia. Unless treated with the greatest consideration they go away and complain of being harshly used, and thus often render themselves very obnoxious to the officials of public institutions. In fact, the very routine which is essential to the proper working of an institution chafes these poor mortals, just as ordinary friction, commonly unperceived, becomes almost intolerable when the cuticle is abraded. There are also black specks before the eyes. There is, too, usually some neuralgia, either intercostal or facial, the former being much the most frequent. On enquiring as to the reproductive system, some disturbance is invariably found. Frequently there is an excessive catamenial flow, extending from seven to ten days, with a leucorrhœal drain during the interval. At other times there may be but a slight catamenial show, with a leucorrhœal discharge constant and profuse; or it may be that the patient is suckling, and a stout infant is the cause of the mother's debility. If there be none of those drains present, or the patient denies them, and it is not always easy to decide which of the two is the fact, there will be found an unwonted indulgence in tea, a neurotic poison of no little potency. Not uncommonly there is a strict and honourable discharge of duty, entailing much exhaustion and further demand upon a system sufficiently handicapped without it.

Such are the general features of the anæmic condition when well pronounced in women of the humbler classes. It is rarely so well defined and so marked amidst the more affluent classes, because the full complement of factors for its production is seldom found in those who have not combined an insufficient body-income and an excessive body-expenditure. The one is scarcely sufficient without the other. And it is the recognition of the combination of these two factors which must guide all treatment, if it has to be crowned with success. Very commonly it appears that there

has been a drain upon the system for a considerable time without any very decided results, until from some cause, either physical or psychical, the digestion is disturbed, and then the consequences are readily apparent. As long as the digestion has been pretty good, the drain has been borne fairly well, but as soon as the nutritive supply of the body has been cut down by impairment of the digestive organs, then general systemic failure is the result. This disturbance of the digestive system may be brought about either by a meal of improper food, exposure to cold upsetting the stomach, or by some mental perturbation. However induced, its advent is soon followed by a general breakdown of the powers. It is no longer possible to meet the heavy body-expenditure with the scanty body-income, and systemic impoverishment is the consequence. Disturbance of the nervous system is part of the general condition, and this is most distinctly seen where tea is resorted to in order to stimulate the exhausted powers. When the wearied and starved organism is kept going by stimulating the nervous system, the taxation of that nervous system manifests itself in neurosal affections of various forms. Still more certainly is the nervous system affected if there be coexistent with this dyspepsia any disturbance in the reproductive system. The relation of this last system to the nervous system of women is well known, but it is to be questioned how far this intimate association is sufficiently appreciated in ordinary everyday practice. Women themselves do not underrate the importance of disturbances in their generative organs, but the subject is a delicate one, and if their tentative remarks are unheeded or deliberately disregarded, they do not usually pursue it, and thus the most valuable information is withheld. Indeed, it is sometimes difficult to get women to admit those derangements of the reproductive organs of which they are so conscious. It is certainly impossible to pursue the subject in the presence of a third party, and still more with a crowd of patients or students around. With the student this is unfortunate, as he is not only so deprived of instruction about a matter of vital importance, but from all enquiry, except of the vaguest and most unsatisfactory character,

being avoided, he is educated to systematically neglect a system whose functions are of the highest importance in the economy. Neither can a proper enquiry be conducted before a host of other women. A woman's privacy must be respected even if she be a hospital patient. Women, too, have a most decided objection to all merely tentative questioning about their reproductive organs; though they are ready enough to give their confidence when it is clearly apparent that the questioner knows thoroughly what he is enquiring about. All such interrogation should be conducted with the privacy of the confessional.

These remarks are not quite out of place as a preface to the consideration of those disturbances of the reproductive system which are so important a matter in the bulk of cases of asthenic neurosal affections. Now as to these disturbances themselves, it may be well to first consider the variations of the catamenial flow. The flux may be insufficient, or it may be excessive. Perfect or partial amenorrhœa is frequently merely a piece of economy on the part of the system, which simply cannot afford the expenditure. This is most often seen in the arrested menstruation of growing girls and of phthisical women. When the growth has ceased, or the tubercular condition been got over, then the wonted discharge is re-established; but it is suspended as long as the system is unequal to it. At other times, however, this suspension may be of more serious import. The absence of the discharge carries with it little evil effect. Its presence in excess is a widely different matter, and often exercises the most disastrous influence. Excessive catamenial losses are of different origin, and their causal relationships are of the greatest practical importance in therapeutics.

In adult and middle-aged women profuse catamenial losses are usually the result of a lack of tone in the uterine vessels, or of congestion of the uterus, or of venous dilatation. The weaker the patients become, the more apt are they to become menorrhagic. At other times, especially in girls, in older and involuntary spinsters, or in women living apart from their husbands, and in widows, the profuse flux is

the result of ovarian excitement. There may be a certain fulness in the womb or in either ovary, but this is not essential. This is a much commoner form of menorrhagia than is ordinarily supposed. Amidst the out-patients of metropolitan hospitals this is especially true. It is not uncommon to find girls, even at fifteen, whose catamenial flow extends over five or six days. It is a fact that this is much more common with small than with well-grown girls: and the question may be raised how far this heavy generative expenditure is not the direct cause of the arrested growth. I say this with some confidence, for in addition to the fact of small undersized girls being more commonly the subject of excessive catamenial losses, in two instances where the flux was checked by full doses of bromide of potassium the girls at once commenced to grow, and added greatly to their stature. There may also be present an uncoloured discharge before and after the menstrual flow, which in many cases extends throughout the whole interval. It may be a steady oozing from the inner surface of the uterus and the vagina, a sort of modified catamenial flow, or it may be more pronounced at certain times. It is most commonly the first when associated with menorrhagia. It is more apt to be the latter when there is amenorrhœa, partial or complete, present. It is commonly associated with an enlarged and heavy uterus, and with ovarian fulness. Where there is present ovarian congestion, there is a tendency to periodical discharges of fluid from the glands of Naboth and Duvernay, especially during sleep, when reflex actions show themselves most pronouncedly. These drains are much more frequent than is ordinarily thought, and are the cause of a great deal of nervous depression among women. Women of a highly nervous diathesis suffer much more from these drains than robust women. Not only are these involuntary orgasms more frequent among such women, but they cause more disturbance of the general health in them than in other women. My observations would lead me to say that the condition here described is not rarely found along with commencing phthisis in girls whose family history is good. It is especially apt to be

produced in girls and women who work sewing-machines, and, worst of all, the double treadle-machines, but it is found wherever women labour with machines which involve the use of the feet while in the sitting posture.

Beyond the drain of a highly elaborated fluid and the nervous exhaustion produced by repeated orgasm, for the more irritable and excitable the parts the more readily is orgasm induced, there are other outcomes of uterine disturbance which are injurious to the patient. One of these is the pain under the heart, or intercostal neuralgia, to which these patients are so liable. Like other neuralgiæ, its associations are those of debility and asthenia; but there seems to be in addition to these some special cause why the sixth or seventh intercostal nerves should be the seats of neuralgia when there is uterine disturbance, accompanied by discharges in abnormal quantity from the vagina. Intercostal neuralgia is not confined to cases where there are disturbances in the reproductive organs, but is so ordinarily associated therewith, and persists so stubbornly until these associated disturbances are relieved, that the conviction is forced upon one in an imperative manner, so much so that it is impossible to evade the conclusion. The pain is persistent, but, like all truly neuralgic pain, it varies in intensity, and often there are recurring gusts of unwonted severity. This pain is harassing and exhausting in itself, while it strongly attracts the attention of the patient from its propinquity to the heart, and thus causes further mental distress. It is a curious fact that this pain is so very commonly on the left side and but rarely on the right; while facial neuralgia, certainly the next most common, is much more frequently, in my experience at least, found on the right side. What are the circumstances which determine these localities we cannot say. It is highly improbable that they are determined by what is termed chance.

At other times, but infinitely less frequently, there are motor disturbances, caused by the perturbations in the reproductive system. There is a certain amount of loss of power, especially of the upper extremities, found along with excessive leucorrhœa. My attention was first drawn to this

subject by an article by Clifford Allbutt in the 'British Medical Journal' (1870), and just after its perusal a well-marked case came under my own notice. The hemiplegia came on with a profuse leucorrhœal discharge; it improved as the discharge was checked, and become more pronounced again when the discharge increased. Several other cases have since come under notice, and to the best of my remembrance they were all left-sided, as was the case with Dr. Allbutt's patient.

It becomes obvious, then, that disturbances in the reproductive system are of great importance in the production of nervous maladies where there exists general imperfect nutrition; and that attention to them is absolutely essential to the successful management of the case. There are not only these sensory and motor consequences just mentioned, but also the centric associations to be borne in mind. It has been said before that one of the prominent features of these conditions of debility is vertical headache, accompanied with a sense of misery and with low spirits, or panphobia. These are the signs pathognomonic of cerebral anæmia, as given in the article 'Cerebral Anæmia' in the fourth volume of these Reports (1874). An attempt is there made to show that the posterior lobes are in intimate relation with the systemic sensations, and that they may be anæmic while the rest of the cerebrum is fairly vascular. The posterior lobes are supplied by branches from the basilar artery formed by the fusion of the vertebræ arteries. The vaso-motor nerves of these arteries are derived from the inferior cervical ganglion, which is in close relation with the sympathetic fibres ascending from the abdomen; while the carotid arteries are supplied by vaso-motor branches from the superior cervical ganglion. Consequently it becomes possible to see how there may be great mental depression from some disturbance in the viscera without the functions of the anterior and lateral portions of the cerebrum being involved or impaired. The associations betwixt melancholia and a displaced uterus, or a mass of scybalæ in the colon, are thus rendered intelligible; and it may be by much the same mechanism that this condition of melan-

cholia is linked with uterine disturbances; as is also the palpitation, which is so commonly found along with functional activity of the generative organs in women, or with some prolapsus of the womb. In these latter cases abstention from erotic indulgence, or the replacement of the womb, gives relief from the palpitation. The irritation in the pelvic organs is conducted by sympathetic fibres to the ganglia of the heart, or the arteries of the occipital lobes; and palpitation is induced in the one case, while contraction of the arteries, leading to local anæmia of the occipital lobes, with its sense of misery and depression, occurs in the other.

After such a survey of the pathological conditions found along with cerebral anæmia and other disturbances of the nervous system of an asthenic character, it becomes possible to construct a therapeutic plan which will produce a decided improvement in the complex condition. The first salient point to be attended to is obviously the combination of causal factors of this state of imperfect nutrition of the nervous system. It is clear that there must be some retrenchment of the body expenditure brought about, as well as improvement in the body income attained. The outgoings must be regulated as well as the incomings attended to. It is of no use to give such a patient milk, beef tea, and tonics, if the drain is permitted to go on unchecked; it would be as rational to pour milk into a sieve. The first point to be attended to is to arrest all drains upon the system. This is essential, for with the coexistent dyspepsia and enfeeblement of the assimilative processes it is simply impossible to increase the body income until it is sufficient to meet alike the wants of the organism and the waste of the drains. The income of the system is limited, no matter how digestible the food, or how easily assimilable, and consequently the greatest economy must be practised if improvement is to be achieved. Without such rigid economy it will be next to impossible to inaugurate any improvement. Consequently, if we find that there is menorrhagia present, it must be checked by means appropriate to its causal associations. If it be due to lack of tone in the vessels of the uterus and its appendages, then tonics with astringents

are indicated. Quinine with dilute sulphuric acid and the extract of ergot may be administered during the day, while a pill of sulphate of copper and opium may be given at bedtime. Others prefer to give the ergot with the perchloride of iron. Much will depend upon the peculiarities of the case; if blood is made rapidly during the interval, and then lost profusely in the catamenial flux—a common condition—then it may be as well to omit the iron. The rapid blood formation causes an increased tension in the vessels, and when the vascular turgescence of the catamenial period comes on the loss is very great. Here it is well to check the loss rather than to increase the blood formation. In other cases where the blood formation is imperfect the addition of the iron is desirable. There is one point of considerable moment to be attended to in checking drains by the internal administration of astringents, and that is their liability to lock up the bowels—an undesirable and unsought, but unfortunately unavoidable, outcome of their action. This can be averted by combining with them the sulphate of magnesia, which is an astringent everywhere except to the intestinal canal. In dram or half-dram doses it adds to the efficiency of the astringent mixtures, while it keeps the bowels open; the last a not unimportant matter, as straining at stool squeezes the blood out of the turgid uterus. In addition to these measures, it is well that the patient should take everything cold during the flux, as hot fluids increase the discharge. If, however, the feet are cold, it will be found that all efforts to keep down the discharge are futile, or nearly so. Under these circumstances warming the feet, by any means soever, not even excepting the hot bottle, will be found of service. If the menorrhagia be found to be associated with erotic excitement, especially when ungratified, full doses of bromide of potassium or ammonium are indicated. These agents, with free doses of alkaline cathartics and aloes, produce quiescence in the pelvic viscera, and give relief. Aloes in small quantities tends to produce fulness in the hæmorrhoidal vessels and their inosculations, but in full doses depletes them; and so is useful alike in amenorrhœa and menorrhagia,

according to the dose. In all cases the use of astringent and tonic injections, by means of syringes and douches, is desirable, the nozzle being preferable to the old bulky syringe for obvious reasons.

Having thus attended to the outgoings, it becomes possible to improve the incomings in a practical manner. If there be any irritability in the stomach it is well to first allay this by bismuth and calumba, together with a suitable dietary. Such a plan might be continued during the interval while the astringent mixtures are resorted to during the time of the flux. When the digestive organs are once more acting efficiently, then the body income can be improved, and tonics and iron may be given freely; the local use of astringents being continued for a considerable period. In the selection of these bitters and chalybeates it will be found not to be a matter of indifference which form is chosen. It is a matter of fact that the lighter preparations, as the ammonia-citrate of iron and calumbæ, or the citrate of iron and quinine, can often be taken with advantage when the muriate of iron will not be tolerated. After a certain progress has been made, then the sulphate of quinine and the persalts of iron may be taken with good effects. In some patients quinine produces intolerable headache, so much so that the patient cannot continue it. Here it is well to give it in hydrobromic acid, or as the bromide of quinine. By such means the headache can usually be avoided. If the mixture of quinine and iron is felt to be too heating, the addition of a little sulphate of magnesia will be found to be advantageous. In most cases of convalescence after asthenia strychnia will be found a useful adjunct to the other medicinal agents. Phosphorus is an agent which is rapidly rising into favour in the treatment of conditions of nervous adynamy. It can be conveniently prescribed in pills.

In addition to these measures it is well to see that the dietary is liberal, and contains a sufficiency of hydrocarbons. These are more readily digestible than albuminoid foods, of which only small quantities are absolutely essential. As soon as fat in any of its forms can be taken, it is well to insist upon its forming a part of the dietary, as it is a valu-

able agent in the treatment of all forms of neuralgia. In all cases tea ought to be prohibited absolutely, and cocoa and milk substituted for it. The distinct aim is to improve the body-nutrition by every available means—to overlook no point. In persons of advanced life neuralgia and the vertical headache, with despondency or panphobia, are often associated with conditions of lithiasis, where there is too much nitrogenised waste in the blood, In such persons the dietary and measures given above are suitable, but they must be combined with alkalies, as potash or lithia, due attention being requisite to the proper depuration of the blood. Such are some of the varied associations of asthenic conditions of the nervous system which it is well to keep in mind in the therapeutic management of these states.

NOTE.—A patient recently wrote me the following note: ‘The first symptom in the head was from a shock, seeing suddenly a cousin who was out of her mind for a time at the change of life. It seemed to shake my nerves. I had to attend upon her six weeks. I have a great pressure at the top of my head: it seems like a sheet drawn down upon it. When that goes off I feel light-headed. The moving about makes me feel as if I should rush against a door or post. Suffered from the whites since fifteen, not quite so much the last twelve months. Memory bad. The least bustle causes confusion. Cannot now read or write much, being so bewildered.’ This speaks for itself.

ON EPILEPSIES AND ON THE AFTER EFFECTS OF EPILEPTIC DISCHARGES.

(TODD AND ROBERTSON'S HYPOTHESIS.)

BY J. HUGHLINGS JACKSON, M.D., F.R.C.P.¹

IN the article in Vol. III. of these Reports ('The Anatomical, Physiological, and Pathological Investigation of Epilepsies') I spoke almost solely of the *direct* effects of epileptic discharges. It is very important, however, to study carefully the After Effects of these discharges—the condition of the patient when the discharge has ceased. I am not now speaking of what is commonly called the inter-paroxysmal condition, but of the temporary state of the patient immediately after the paroxysm—which will be called the post-paroxysmal condition. This has not, so far as I know, been studied methodically. We have descriptions of loss of con-

¹ I find that the explanation which I thought I was the first to give of the effects of strong epileptic discharges in producing temporary paralysis has been given before, as the subjoined quotation shows. I fully acknowledge the priority of Todd and Alexander Robertson by giving the quotation and by placing their names in the title of the article. Thus I may proceed in the text in my own way. This article was written before I came across the following or before it attracted my attention:—'But I am inclined to think that the late Dr. Todd was correct in supposing that severe and protracted convulsions may themselves in some instances be causative of palsy of a few hours' or days' duration, through simply the exhausting influence exerted on the cells of the central ganglia without much, if any, appreciable change of tissue. This explanation is especially applicable to some cases of hemiplegia following epilepsy in which the paralysis passes away in a few days.'—Alexander Robertson, 'Ed. Med. Journal,' December, 1869.

sciousness or coma remaining after severe epileptic seizures, but the physical phenomena have not been carefully analysed even in these cases. We have, too, accounts of mania after epileptic seizures ('epileptic mania'), but I submit that the accepted interpretation of this important sequence is not in accordance with facts. The after-effects of epileptic discharges of subordinate cerebral centres causing limited convulsive seizures, have commonly received least attention. Yet these cases are by far the most profitable for investigation of the after effects of excessive nervous discharges (epileptic discharges) in general. If we arrive at an explanation of the causation of post-epileptic hemiplegia, we shall then more easily explain post-epileptic loss of consciousness with mania (Epileptic Mania). The clinical nomenclature is confusing: thus the mania in Epileptic Mania is not a thing analogous to hemiplegia in Epileptic Hemiplegia; for besides other obvious differences, the former is a positive condition, the latter is a negative one. Some preliminary remarks on epilepsy are needed.

An epileptic discharge was defined (Vol. III. p. 331) as an occasional, sudden, excessive, rapid, and local discharge of some *part* of the cerebral hemisphere—a discharge of some *part* of the cortex which has become highly unstable. There is what I call a 'discharging lesion.' These lesions are, as above said, supposed to be always local, notwithstanding that their discharge, when in the highest centres (as in so-called genuine Epilepsy), may produce convulsion of all parts of the body nearly contemporaneously. As above defined, the term Epilepsy is not the name for any one grouping of symptoms, but for any set of symptoms whatever presented paroxysmally from discharge of some *part* of the cerebral cortex. Whether consciousness be lost or not matters nothing for this definition.

What is to come will be unintelligible unless the reader bears in mind—

(1) That it is assumed that all nervous centres, from the lowest spinal centres to the very highest centres (the substrata of consciousness), are made up of nothing else than nervous arrangements representing impressions and move-

ments. The fact that movements are not produced by slight galvanic or faradaic currents applied to the cortex beyond Hitzig and Ferrier's Region, does not disprove this. I do not see of what other 'materials' the rest of the brain can be made. The term 'impression' includes all cases where a peripheral effect (skin, tissue, or viscera) disturbs a nervous centre, and the term 'movement' is used in an unusually extended sense, to cover not only effects produced by nerve centres on muscles (including arterial coats, muscular fibres of intestine, &c.), but on glands and effects by inhibitory nerves.

(2) That states of consciousness, although always parallel with, are utterly different from, nervous states, which in the higher (the cerebral) centres are, as in the lower, concerned with impressions and movements. It is not said that mind is made up of nervous arrangements for impressions and movements, but that the substrata of mind are thus constituted. Hence we do not say that any kinds of mental state *arise from* nervous discharges, but that they *occur during* nervous discharges.

Partly to enforce these remarks, I will repeat, but I hope with more precision, what I said (Vol. III. p. 331) as to the kind of distinction I make betwixt cases of epilepsy where there is loss of consciousness and cases where there is not. Let me remark first that the presence or absence of consciousness in an epileptic paroxysm is not even the *empirical* distinction which I make betwixt different kinds of epilepsy. The empirical distinction is into cases where consciousness is lost first of all in the paroxysm, soon after the onset, late, or not at all. The distinction scientifically is that consciousness is lost at the onset of the paroxysm, or almost at the very first, when the discharge *begins in the very highest nervous centres*, these centres being the substrata of consciousness. Consciousness is lost late when the discharge begins in a subordinate centre—in some part of Hitzig and Ferrier's Region, for example. In epileptic discharge of these centres consciousness may not be lost at all; all depends on the momentum of the discharge, and therefore on how far it spreads. How it is that consciousness is lost during epileptic dis-

charges of some *part* only of the highest cerebral centres will be explained later.

In very slight epileptic discharges, even of the highest centres, there may be only slight defect of consciousness, with only slight confusion of thought. Briefly, whether consciousness be lost or not depends on the *seat* of the discharging lesion and on the momentum of the discharge.

Another thing (a corollary from the foregoing) never to be forgotten is that the affection of consciousness is not a 'symptom' comparable with any kind of abnormal physical state. One reason for this remark is that in my former article (Vol. III. p. 331) I wrote: 'Loss of consciousness is not an utterly different thing from other symptoms.' This was a very blundering statement, but the context showed clearly, I hope, that what was meant was that cases of epilepsy, with loss of consciousness at the onset, depend on discharge of sensori-*motor* centres, just as do cases of epilepsy (or, as they are commonly called, epileptiform seizures) in which there is no affection of consciousness, the *difference* being not in the kind of constitution of the centres, but in their degrees of evolution. The following statements are, I hope, free from such blunders.

Suppose that from an epileptic discharge of a subordinate motor centre we have convulsion of the arm; we do not compare this with loss of consciousness in a case of *le petit mal*, where there is epileptic discharge of nervous arrangements in the highest centres. We compare the spasm of the arm from discharge of the subordinate cerebral centre with spasm of arteries (as signified by facial pallor), of the eyes, hands, arrest of respiration, &c., from discharge of the highest cerebral centres. That consciousness ceases during the latter discharge, and not during the former, is not the thing of moment in an anatomico-physiological enquiry.

According as the seat of the discharging lesion varies the symptoms of the paroxysm vary. And since the cerebral centres—even the highest of them—represent or re-represent all parts of the body, we have all kinds of symptoms in the paroxysms from differently seated discharging lesions. Let us give a rough list of some of the more important of

them:—Increased flow of saliva; pallor of face; shivering, with *sensation* of cold; arrest of respiration, with *sensation of suffocation*; *coloured vision*; *noises in the ears*; *nausea* (and other less definite *sensations* referred to the epigastrium); movements of the eyes with *vertigo*; convulsion of the limbs, &c. The words in italics are names of mental states (sensations); the physical states corresponding to them are of course meant and are in some of the cases mentioned. Which of these symptoms occur, or which preponderate, or in what order any of them occur in coexistence or in sequence, depends on the seat of the discharging lesion and on the momentum of the discharge. In other words, every epilepsy is a development, but a brutal development, of the functions (1) of some *part* of the cerebral cortex, the cells of which part by some pathological process have become highly unstable; and (2) especially when the momentum of the discharge is great, of collateral and lower healthy centres. From this it follows that there is, scientifically speaking, no entity to be called Epilepsy; but innumerable different epilepsies as there are innumerable seats of discharging lesions. And as the first symptom in the paroxysm is the first effect of the discharge of the centre unstable, any two paroxysms *beginning* differently will differ throughout, however little. For practical purposes, it is convenient to have arbitrary types—definitions by type—but these are of no use in scientific investigations.

Recent experiments seem to show that what has been called ‘menial’ work is part of the work of the cortex cerebri. ‘A relationship has been observed between the brain surface and the secretion of saliva, the beat of the heart, the action of vaso-motor nerves, and other organic functions; but on these points the results of various observers are by no means constant.’ (Foster’s ‘Physiology,’ p. 441.) It would be a very remarkable thing if the organic functions *were not* represented in the cortex cerebri. It would be very remarkable if the highest centres, the sub-strata of consciousness, did not represent the whole organism, the tissues, viscera, arterial system, and muscles. The physical phenomena occurring with transient loss of consciousness,

in cases of *le petit mal*, would be unintelligible if the 'organic functions' were not represented in the very highest centres. It would be most remarkable if the heart were not represented in every unit of the very highest centres.

For a fit to begin with loss of consciousness is for the epileptic discharge to begin in the highest centres (see p. 268). As regards two symptoms showing disturbance of 'organic functions,' increased flow of saliva, and deep facial pallor, it is to be observed that they will occur from the very slightest epileptic discharges of the highest centres; that is to say, in cases of *le petit mal*. Herpin writes:—'*L'accumulation de la salive dans la bouche et dans la gorge est un des signes les plus constants des accès d'épilepsie. On le retrouve même dans les simples vertiges.*'—'Epilepsy,' p. 432.

Crichton-Browne (these Reports, Vol. III. p. 157) writes: '... among the phenomena of the fit itself, pallor of the face is perhaps the earliest and most constant.' Speaking of some cases of *le petit mal*, he says (op. cit.):—'... this pallor is *the only* outward sign of the fit, and corresponds with the momentary unconsciousness or loss of perception and volition in which it consists.'

It is to be noted, too, that emission of urine and fæces often occur in slight cases of epilepsy due to epileptic discharges beginning in the very highest centres—*le petit mal*. Herpin, op. cit. p. 433, writes:—'*Vomissements, selles, vents, ne sont montrés au milieu des accès que dans des crises fort légères.*' My own opinion is, however, that these symptoms do not occur *in*, but *after*, the paroxysm; they are too co-ordinated movements to result directly from epileptic discharges; there is, I think, a duplex condition (1) negatively, loss of control; (2) positively, increased activity of healthy lower centres. Nevertheless, the association or sequence, is very significant.

Equally unintelligible would be the occurrence of the bodily manifestations observed in normal emotion, or in the abnormal¹ emotion of hysteria. These manifestations are

¹ I believe, however, that there is a duplex condition in emotional states—negative (loss of control) and positive (over-activity of lower centres). I have seen somewhere, but where I cannot now remember, a remark on the strangeness that

exhibited in 'all parts of the moving system, voluntary and involuntary; while an important series of effects are produced on the glands and viscera—the stomach, lungs, heart, kidneys, skin, together with the sexual and mammary organs.'—Bain, 'The Emotions and the Will,' p. 4.

I do not found these statements, however, on any recent experiments, properly so called, but on the experiments of disease. In the 'Medical Mirror,' October 1869, I write, except for a few verbal alterations, as follows:—'We have now, then, to add to the constitution of the units of the cerebrum nerve fibres to the heart, vessels, and viscera, or rather probably to regions of the Sympathetic system, from which these parts are supplied. The inference we have now arrived at is that the units of the cerebral hemisphere (*in the region of the corpus striatum, at least*) represent potentially the whole processes of the body. If this be so, we can understand how it happens that in cases of epilepsy [beginning by loss of consciousness, i.e. the discharge beginning in the highest nervous arrangements], besides obvious convulsion, we have premonitory shivering, pallor of face, and increased flow of saliva, and in some cases vomiting. Thus, too, we see how it is that emotional manifestations accompany intellectual phenomena. Emotional manifestations are wide and yet temporary bodily states, and we have seen that the heart, arteries, and viscera, as well as the large muscles of the body, are represented in the units of the cerebrum.' I should now omit the words italicised, believing, as repeatedly stated, that the substrata of consciousness, the highest cerebral centres, as well as those in the region of the middle cerebral artery, represent the whole body. All the work of the cerebral cortex is 'menial' if the secretion of saliva or the movement of a limb is menial work. During the highest kind of ideation there is menial work; for when we think of an object there is, on the physical side, an excitation of nerve

experimental irritation of the 'centre' for an animal's tail should be the same centre as that for the secretion of saliva. Although I do not believe in such abrupt localisations, I see nothing remarkable in the association; the salivary glands will be in vigorous action when a lamb is sucking; its tail moves about in a ludicrous manner during that operation. Both parts are in action together during one emotional state.

cells and fibres representing retinal impressions and ocular movements.

These assertions would be strange indeed if they are taken to mean that nervous arrangements representing muscles, viscera, &c., constitute consciousness; but that inference has been repudiated. It is only said that they constitute the anatomical substrata of consciousness. No one knows why energising of any cells and fibres representing (1) muscles, tissues, viscera, &c.; or of cells and fibres representing (2) things in general; or of cells and fibres representing (3) nothing at all; or of cells and fibres representing (4) nothing in particular, is attended by any sort of psychical state. But although active nervous states are not psychical states, there is parallelism, and what we have to do in a medical enquiry is to discover the anatomy and physiology of the various nervous states *that go along with* various psychical states; and we have to note the *physical* manifestations when consciousness ceases during epileptic discharges. And those physicians who believe that nervous states and physical states are one and the same thing are as much bound to take note of the physical manifestations which occur from discharges of 'ideational centres' as those are who hold the commonly-received doctrine that psychical states are utterly different from, although they always occur along with, nervous states.

From the above remarks it will be inferred that by highest centres we do not mean geographically highest, but anatomically highest. The highest centres are those which represent the most complex and most numerous different co-ordinations of impressions and movements of all parts of the body.

The symptoms of an epileptic paroxysm may be motor (convulsion), or there may be evidence of discharge of sensory centres. The hypothesis, I hold, is that the anterior part of the brain is *chiefly* motor, the posterior *chiefly* sensory. The evidence as to epileptic discharges of sensory centres obviously can only be indirect. We see nothing: the patient can only tell us of the mental states which occur along with them; he tells us, for example, that he has numbness or coloured vision. From this kind of evidence

we *infer* discharges of sensory centres. The coloured vision, the numbness, &c., are crude psychical states; the corresponding physical states are the nervous discharges of sensory centres. Moreover, the patient can only tell us of crude psychical states occurring with the beginning of the epileptic discharges of sensory centres, although the discharge of sensory centres may doubtless continue long after his consciousness is lost. Hence the evidence as to discharges of sensory centres is not only indirect—it is also limited. However, in some cases of Migraine, which in my nomenclature are Epilepsies, the development of sensations during discharges of sensory centres is slow and deliberate, and consciousness is not lost. In these cases states of sensory centres corresponding to the sensations may be studied; we can, however, only infer them. But motor symptoms, *e.g.*, convulsion, are far more easily studied. Here again we must insist on the distinction betwixt psychology and the anatomy and physiology of the nervous system. It is a most unfortunate thing that the word ‘sensation,’ the name of a state of consciousness, is of the same derivation as ‘sensory,’ the name given to afferent nerves and to centres to which afferent nerves go: it fosters the confusion that a physical state in a sensory centre *is* a sensation. ‘It is usual, indeed, to speak of sensations as states of body, not of mind. But this is the common confusion of giving one and the same name to a phenomenon and to the proximate cause or conditions of the phenomenon. The immediate antecedent of a sensation is a state of body, but the sensation itself is a state of mind.’—*Mill’s Logic*, 8th ed. vol. ii. p. 436. The fact is that ‘sensations’ (which are not physical states of any kind) attend energising of motor as well as of sensory centres. Using here mixed psychological and anatomico-physiological language (and legitimately, because we are at this moment speaking both of psychical and physical states), we may say that there are not only sensory-sensations, but motor-sensations. In healthy psychico-physical operations they are conjoined. Thus, whenever we see an object, we have the sensory-sensation of colour, and the motor-sensation of shape (relations of positions). ‘It may be said that *all* the senses

are not physically sensori-motor. I think they are. Thus the auditory nerve has evidently (as the symptoms of Meniere's Disease show, and anatomy confirms) very extensive motor associations. I have tried to show this.'—'Med. Times and Gazette,' Aug. 7, 1875.

In disease we may have more nearly pure sensory or motor manifestations than we can have in health, because the sensory and motor elements are for the most part geographically separate (p. 273), although physiologically connected. Taking for comparison a morbid sensory-sensation and a morbid motor-sensation from the same class—the visual—as in the illustration just given, we have in one case of epilepsy almost purely the motor-sensation of vertigo (a sensation during extreme energising of ocular motor centres), and in another case almost purely the sensory-sensation of colour (a sensation during energising of centres representing retinal impressions). But I do not think we ever have even in disease absolutely pure sensory-sensations or absolutely pure motor-sensations. Thus, in the ocular phenomena of Migraine, there is often a zigzag outline, and with epileptic spasm of the hand there is commonly abnormal sensation referred to the skin. The zigzag or fortification outline implies excitation of motor elements; of those I suppose which serve in giving us ideas (symbolically of tactual ideas) of roughness (minute shapes), and which are so immediately, inevitably, and deeply organised with their corresponding retinal impressions that roughness and colour seem to be one sensation. Then the 'balls of fire' at the onset of some epileptic seizures must be of some shape; to say that by the constitution of the mind the patient is obliged to think of them as of some shape is true. But what is the physical side of this obligation?¹ It is excitation of centres for ocular movements.

Since the cerebrum is the 'organ of mind,' it might be supposed that *elaborate* mental symptoms would occur from,

¹ By the 'constitution of his mind,' 'by an act of volition,' a Migrainous patient of mine can vary the size of his ocular spectre, but to do so he must, at the same time, alter his accommodation; the spectre is about four feet across when he looks at the window, a distance of eight or nine feet, and the size of a sixpence when he looks at the page of a book, ordinary reading distance.

or rather *occur during*, epileptic discharges, if these be, as I suppose, discharges of the cerebral cortex.

As will be insisted on later, elaborate mental states do not occur during an epileptic discharge—as a *direct* result of that discharge, we mean. During epileptic discharges we have only crude mental states, such as ‘balls of fire before the eyes,’ noises in the ear, &c., and then only at the onset of the discharge, when it is presumably *comparatively* slight and slow. Yet these incipient discharges will be far stronger than those of health if we may suppose the strong expressions of the patients (‘balls of fire,’ &c.) imply vividness of sensation. It is possible to show by experiment that we can have more vivid sensations of colour ‘subjectively’ than any that are caused by the presentation of outward objects. (See ‘Helmholtz’s Popular Scientific Lectures,’ p. 256.) But this admission does not involve the admission that we have such *elaborate* mental states as spectral illusions or ‘voices.’

As was urged in the article on ‘Temporary Mental Disorders after Epileptic Paroxysms,’ Vol. V. of these Reports, elaborate mental states occur only during activity of centres, which, except for slight over-activity, slightly increased discharges ‘from loss of control,’ *are healthy*. There is a duplex condition; negatively loss of function of the highest, and positively increased function of lower centres. Elaborate mental states imply special, and complex co-ordinations in co-existence and succession of vast numbers of sensations, which are to the elaborate states as raw material to elaborate structure. Mere increase in vividness of one or of many sensations is not an elaborate mental state.

Except at the onset of an epileptic seizure when there are crude mental states, such as coloured vision there occur *no* *psychical* states of any kind *during*, but only *physical effects from*, the excessive local discharge, whatever the part of the ‘organ of mind’ may be in which the ‘discharging lesion’ is seated.

We have in all discharging lesions of any parts of the organ of mind a brutal development of the impressions and movements represented in the part discharged, and no elaborate mental states attend these developments. The elaborate

mental states called intellectual auræ (spectral illusions, 'voices,' &c.) do not occur during epileptic discharges. They occur, I think, during over-activity (slightly increased discharges) of lower nervous arrangements not yet reached by the epileptic discharge. A spectral face is a mental state infinitely more elaborate than 'balls of fire.' In some cases of epilepsy we have the double warning of (1) coloured vision, so to speak, 'turning into' (2) spectral visions of faces. Abandoning this misleading metaphor, I think the relation is that whilst the coloured vision is the result of, or rather occurs *during*, the onset of the epileptic discharge, the spectral face occurs *during* the springing into activity of the next lower nervous arrangements, which are healthy, except for excitement from loss of their higher 'controlling' nervous arrangements put out of use by the epileptic discharge. But the epileptic discharge, mostly, but not always—for the fit may stop at the stage of the spectral faces—soon involves also the lower nervous arrangements, and, maybe, produces universal convulsion. Similarly for warnings of noise in the ear, followed by 'voices.'¹

We shall consider in this paper, to begin with, cases of epileptic discharge of cerebral motor centres, that is, cases of convulsion. We shall select cases in which the discharging lesion is in subordinate motor centres; we say *subordinate*, meaning thereby centres in Hitzig and Ferrier's region, because, as we have said, we believe all, even the very highest, nervous centres to be made up of nervous arrangements representing impressions and movements. There is in these cases excessive local movement, i.e. local spasm, for a short

¹ The term 'voices' is inaccurate when applied to cases in which the patient has the delusion, or illusion, or hallucination that someone is speaking to him. The so-called 'voices' imply activity of the sensori-motor substrata of words, and of those substrata which are in health concerned in *receiving* speech; these substrata lie chiefly, I think, on the right side of the brain. This explanation is not a verbal one, for the ordinary opinion seems to be that auditory centres or sensory perceptive centres only are concerned. Again, the ordinary opinion is that speech is a single linear process—that an automatic reproduction of words does not precede speech. The view I take is that speech is the end of a process of 'verbalising,' of which the automatic reproduction of words is a necessary first part. Speaking generally, all nervous processes are dual, and there is a corresponding duality in all mental processes; very evidently in perception, as cases of simple delirium (imperception) show.

time. When the discharge has ceased and the convulsion is over, we very often find paralysis of the parts which were convulsed—or, rather, which were first and most convulsed—of those parts first and most discharged upon. For example, after a convulsion beginning unilaterally, there is very often hemiplegia. Hemiplegia so occurring was called Epileptic Hemiplegia by Dr. Todd. There are other after-effects of epileptic discharges of other subordinate centres. There is an ‘epileptic aphasia.’ This and the local palsies are the after-effects of excessive discharges of subordinate motor centres (in Hitzig and Ferrier’s region). Further, after severe epileptic discharges, beginning in the very highest centres, there is a very wide physical prostration: this is *attended by* loss of consciousness, or rather *during this state* consciousness ceases. Or there is coma, according to degree of the discharge, and subsequent prostration. Although empirically we may compare the ‘loss of consciousness,’ with hemiplegia, it is obvious, from many prior statements, that the condition which we scientifically compare with epileptic hemiplegia is not loss of consciousness or coma, but the prostration of more or less of the organism, *during which consciousness ceases*. Similarly epileptic aphasia should be post-epileptic exhaustion of more or less of those highest articulatory nervous arrangements which are not words, but the substrata of words, and the substrata of those words which serve in Speech. We shall, however, use the empirical expressions until we deal particularly with the states referred to.

It is better to speak of post-epileptic hemiplegia, post-epileptic aphasia, and post-epileptic loss of consciousness. For there are two opposite conditions of nerve centres, under which the use of parts is lost. We have already stated fully what, for another purpose, we now state briefly again, and with some new illustrations more directly to our purpose. The use of parts is lost *during* those excessive discharges which we call epileptic of their centres. *During* convulsion of the arm, as much as during paralysis of it, the patient has no use of that limb. This seems a pedantic refinement. But the principle is gravely important when applied to cases of aphasia and to cases of loss of consciousness. During the

epileptic discharge of the centres which are the substrata of speech, there is loss of speech; *during* the excessive discharge of the centres which are the substrata of consciousness, there is loss of consciousness. This sounds contradictory; it seems like saying, that during 'excessive function' there is 'loss of function.' It is so in effect. It is certainly true that what is erroneously called '*mental function*' ceases *during* excessive discharges; and *from* excessive discharge of nervous centres the *proper use* of the parts excessively innervated is lost. It is evident enough in some cases; the Migrainous patient can see nothing in the part of the field covered by his spectre; his visual nervous centres are otherwise engaged.

All active bodily phenomena in health imply nervous discharges; when we think of an absent object, as well as when we grasp it or see it, there is a nervous discharge. During the most refined emotional state, without obvious outward change, there are nervous discharges, as well as when we laugh or shed tears. But in epilepsy the discharges are excessive; during the *rapid* discharges of *vast numbers* of the elements of the substrata of consciousness there could arise no conscious states. *Time*¹ *is required for consciousness.* What ensues on *excessive* discharge of a part of the brain is a development of the impressions and movements represented in that part; there is not a development of mental states, but only a brutal development of the functions of the *substrata* of mental states; during this consciousness ceases. Thus (speaking of the motor elements only), in epileptic discharges of centres for visual ideas, we should have spasm of the muscles of the eyeballs; in epileptic discharges of the centres for tactual ideas we should have spasm of the hand; in epileptic discharges of the centre for speech we have spasm of the articulatory muscles. In epileptic discharges of the highest centres, which are the substrata of consciousness, we have implica-

¹ 'In the internal perception of a series of mental operations, a certain time, a certain duration, is necessary for the smallest section of continuous energy to which consciousness is competent. Some minimum of time must be admitted as the condition of consciousness.' Sir W. Hamilton's '*Lectures*,' i. 369, quoted in 'Mill on Hamilton,' p. 277.

tion of the whole organism. But no ideas of objects, of words, nor any kind of states of consciousness, occur during these *excessive* discharges excepting crude sensations at the onset.

We now speak of post-epileptic loss of function, and we begin with the simplest cases—post-epileptic loss of function of subordinate cerebral, and probably often of still lower centres, for movements of the limbs.

In most cases there is, I think, temporary exhaustion, or, more technically speaking, temporary loss of *function* of the nervous centres in which the discharge began, and of centres through which the nerve-currents developed passed. In an article in Vol. III. of these Reports I urged that there were two great divisions of functional changes:—(1) those of *loss* of function; and (2) those of *over* function. As there stated, the term functional is not used for slightness of pathological change, nor applied clinically to cases when symptoms are trifling and transitory. It is, when used medically, limited to the description of the morbid alterations of *the proper function of nerve tissue*, which is to ‘store up’ and ‘expend’ energy.¹ We must not confound functional states (abnormal physiological conditions) with the morbid nutritive processes leading to them (pathological conditions). In cases of convulsion followed by temporary hemiplegia there are both functional states. There is first over-function (excessive discharge signified by the convulsion), and next, loss of function (exhaustion of nerve tissue signified by the hemiplegia), the after-effect of the excessive discharge. There is in these cases a destruction of the function of nerve fibres, not a destruction of fibres. That is to say, there is no breaking up of structure, but the fibres cease to be nerve fibres in effect so long as they are unable to ‘carry’ nerve-currents. The destruction of function after an epileptic discharge is only temporary. But however slight and however recoverable, the physiological condition is, so long as it lasts,

¹ I would here remark that I use such expressions as ‘store up energy’ in the sense that the nerve cells are nourished by nutrient fluids containing potential energy; such expressions as ‘currents passing,’ &c., are used conventionally, and not as implying any particular hypothesis.

fundamentally the same as when nerve-fibres are broken up. It is a most unfortunate error to confound losses of function with the pathological processes leading to these losses of function. Increased excitability of the medulla oblongata is said by most authorities to be the *pathology* of epilepsy; but increased excitability is an *abnormal physiological* condition. The pathological question is, by what disorder of the nutritive process do nerve cells become unstable or excitable?

I do not say, however, that there may not be coarse disease of the brain in these cases, and thus physical destruction of cells and fibres. In post-epileptic hemiplegia there very often is tumour. All I say is that the paralysis is not necessarily the result of that destruction. In some of these cases we discover post-mortem a tumour, involving convolutions near to the corpus striatum. The tumour *has* destroyed more or less of the cortex; this is admitted. But it would be most misleading to conclude in such a case that the tumour has necessarily 'caused' paralysis when that paralysis followed convulsion. Certainly it does 'cause' it, but not always directly. It does not cause it because it has *destroyed* so much of the brain. It causes it in a doubly indirect way. As a 'foreign body' it is the initiator of abnormally increased nutritive changes (pathological process) from which results the abnormal physiological condition of over-function—instability of neighbouring grey matter (instability of cells constituting a 'discharging lesion'). The unstable grey matter discharges excessively and suddenly, and the final step is supposed to be exhaustion of the part discharged, and, more important still, of lower centres out of which the centre (which has become unstable) is evolved.

We have already ('On Evolution of Nervous Centres,' Vol. III. p. 362) drawn attention to the fact that destruction of parts of the convolutions in the neighbourhood of the corpus striatum may produce no obvious paralysis, whilst the epileptic discharge of those parts would produce severe convulsion. These facts show that movements are largely represented in parts, destruction¹ of which parts causes no

¹ Referring to previous remarks of mine to this effect ('Brit. Med. Journal,

permanent loss of movement. The seeming discrepancy is explainable on the Principle of Compensation. The higher a nervous centre the more tolerable is a given 'quantity' of destruction, and the more intolerable an equal 'quantity' of instability. Another thing, well brought out by Charcot, Lepine, and Landouzy, is that, so far as the difference betwixt the corpus striatum and the convolutions in Hitzig and Ferrier's region are concerned, the more limited in range is the paralysis from destruction, the higher the centre. This is not, I think, because the centre destroyed does not represent, or rather re-represent, *all* the movements represented in the corpus striatum, but because it represents some very specially, and represents others generally, that is in common with all neighbouring centres. Thus, to take an arbitrary and limited illustration, supposing one centre in Hitzig and Ferrier's regions to represent specially the hand, another specially the face, another the foot, I should believe that each one of them represented all the movements of the chest.

I put forward the doctrine which I now call the Principle of Compensation in an article in the 'Medical Times and Gazette,' December 21, 1867. I mention this particularly, because it has been implied in some quarters that I am not aware of the 'suppleance' of one part of the hemisphere for another. Since, in the article referred to, I write, 'So the speculation is that, although each movement *is everywhere represented*, there are points where particular movements are specially represented,' it would have been less erroneous to assert that I have applied the principle too audaciously. (See also the quotation given from the 'Medical Mirror' at p. 272 of this article and illustrations later on in this article.)

May 10, 1873) Dr. Day of Stafford says ('St. Andrews' Medical Graduates Transactions,' vol. vi.) that I endeavour 'to prove, by *speculation*, that although the movements will take place even when the parts in which the function is believed to reside are destroyed, yet, in spite of, or rather notwithstanding this, these parts *do* represent the special movements referred to. Thus making it appear that the movements can take place either with or without this portion of cerebral substance, or, in other words, that these particular anatomical conditions are, for the purpose in question, *supernumerary*.' As stated in the paper Dr. Day is criticising, the explanation is that there is in other centres a representation, although a less special one, of the movements most specially represented in the centre destroyed.

The tumour does not produce the hemiplegia because it has destroyed so much. It destroys slowly. There is often no paralysis from *such* slow destruction of convolutions, or no notable paralysis. I say 'no notable paralysis,' because I do not think Compensation is ever absolute. There is evidence of Compensation from experiments on the brains of animals. It has been found that in cats and dogs extirpation of some part of the cortex, which Hitzig and Ferrier have shown to represent a given set of movements, is not followed by permanent loss of those movements. There is temporary paralysis only, after which, as I would express it, there is Compensation by neighbouring centres. I have never believed in what I call abrupt localisations. I do not believe that there is any part, for example, where the movements of the hand are solely represented; but that there are numerous parts where these movements have special or leading representations; there being in each, as the term 'leading' implies, a representation of other parts serving subordinately with the leading movement. I have never acceded to the opinion that speech is to be localised in any one spot, although I do believe most firmly that the region of Broca's convolutions is, so to speak, 'the yellow spot' for speech, as the macula lutea is the centre of greatest acuteness of vision, although the whole retina sees. Even in the highest centres I hold that each of the component units represents the whole organism, although each unit represents it differently from all others, however slight the difference may be betwixt many of them. In the case of tumour, the destruction is very slowly effected, hence often no discoverable palsy. The experimental destruction is rapid, hence decided palsy for a time. The difference of rapidity is an exceedingly important difference. The temporary palsy in the experiments results not from lack of *the part extirpated*, but from *sudden loss of it*.

Nevertheless, I will not deny, as I have formerly done, that permanent paralysis may result from *wide-spread* destruction of certain convolutions, and especially am I inclined to admit it after reading the physiological evidence adduced by Hitzig and Ferrier, and the clinical and pathological

evidence of Charcot, Lepine, and Landouzy. But I do not think that *paralysis after epileptic convulsion* is thus explicable, as I shall try to show.

Let me now formulate the general principle. It is—*‘Parts of the Central Nervous System are temporarily exhausted by Epileptic (that is, excessive) Nervous Discharges.’* Just as after great but healthy exercise there is fatigue of nerve centres and muscles, so, after the outrageously violent ‘exercise’ in a convulsion, there is an excessive fatigue—a fatigue to that degree which we call paralysis. We begin by the simplest problem, that is, with limited convulsions, followed by paralysis of the parts first and most convulsed; in other words, we begin by the consideration of excessive nervous discharges of *subordinate* motor centres followed, according to the hypothesis, by exhaustion of the centre discharged, and of nerve fibres and cells of those lower centres which the current has reached or through which it has passed. We limit ourselves to the after effects of epileptic discharges as these are seen in muscles, ordinarily so-called. In some cases there is, as there is after the nervous discharges of a rigor, a flaccidity of arteries; a paralysis or paresis of their muscles (muscular coat) after their spasm. We speak of the effects seen in muscles of course in order to infer the condition of motor centres for those muscles. Unless we begin by these simple cases, I do not see how we are to find any principle to explain the more difficult cases of post-epileptic aphasia and post-epileptic loss of consciousness accompanying epileptic mania.

There is not always local paralysis, or at any rate *obvious* local paralysis after limited convulsion. Paralysis is found when the discharge has been very severe, its severity being estimated by the severity of the convulsion. In such cases those parts which have been first and most convulsed are temporarily paralysed.

Logically there ought always to be *some* paralysis after every nervous discharge which is in excess of healthy nervous discharge, and no doubt there is. There will at any rate be the primary exhaustion from loss of energy of the unstable cells which constitute the ‘discharging lesion,’ even if no

secondary exhaustion of collateral and subordinate healthy nerve cells and fibres. For even after discharges of health there follows exhaustion, evidently if the discharge be a little above the common; hence fatigue, and in the case of vision the negative after-image following a positive after-image.¹

In cases of epileptic discharge of centres representing retinal elements during which a vivid colour arises, we should expect, on the hypothesis stated in this paper, that the patient would be unable to recognise the colour of objects of that colour when the discharge had ceased; that the central anatomical elements corresponding to it would be much exhausted. I have never tested this. Dr. Alexander Robertson has ('British Medical Journal,' April 18, 1874). 'In this case the subjective reproduction of red as a distinct aura was followed by a temporary inability to recognise that colour for a time.'

Practically speaking, there is no motor paralysis except after *severe* epileptic discharge.

The reader will observe that it is not said that those parts

¹ Many cases in which there really is exhaustion are misinterpreted. As several times mentioned in this article (see p. 271, etc.) a patient's symptomatic condition is often duplex; negative and positive; often the positive symptomatic condition attracts exclusive attention. In some cases it seems to me to be evident that contemporaneously with the loss of function of a centre, there is a rise in activity of the next lower centre. (Tbompson Dickson's 'Principle of Loss of Control.') I shall have very often, for various purposes, to show the wide bearings of this principle. I believe that it applies in the explanation of the occurrence of complementary colours—there is here also a duplex condition negative and positive. To take a particular case: exhaustion of some of the nervous elements which are the substrata of the colour red, is contemporaneous with, or is soon followed by, increased activity of those elements which are the substrata of blue green; such increased activity being a consequence of the exhaustion; there is 'loss of control' on a minute scale. The blue green arises when, the eyes being closed, no light enters the eye, and thus that complementary colour is owing to 'spontaneous' activity of the central substrata for that colour; is not caused by fresh peripheral excitation. Further, I would suggest that the same occurs on a minute scale in ordinary vision; that in seeing any colour, there follows not only trifling exhaustion, or let us use here the less strong word, fatigue, but also a trifling rise in activity of the substrata of all other colours, probably in some order. The obvious objection is that we have no conscious states, no colours corresponding with this hypothetical rising in activity. But we cannot know any one colour except by comparison and contrast with other colours, and hence *à priori*, we should assume that, although there is no vivid consciousness of the other colours, there is some consciousness—a 'sensibility' of them, to use Lewes's term.

which have been convulsed, but that those which have been first and most convulsed, are those left paralysed. The paralysis varies in two ways—in Range and in Degree. In these respects it is the correlative of the convulsion which it follows. Convulsions beginning locally in the hand, face or leg attain all ranges, and are of all degrees of severity; the post-epileptic palsy varies similarly. As to Range there may be palsy of the hand only after a convulsion beginning there and affecting little more than that part, or there may be hemiplegia after a severe and universal convulsion beginning in the hand, and affecting the side it began in most. As to Degrees; after a slight and partial convulsion there may be either only such slight loss of power as prevents a patient picking up a pin, although he can strike a blow, or after a severe convulsion there may be absolute paralysis of an arm, or leg, or there may be as much hemiplegia as a large clot causes. When the fit has been very slight the patient may speak only of numbness, or use less definite expressions, as that ‘the hand feels queer.’ The numbness is, I think, owing to the loss of use of nervous arrangements for innumerable, minute or limited movements, ‘delicate movements’ which, associated with correspondingly minute skin impressions, give us ideas of infinitesimal shapes such as roughness of surface; there is a loss of use of some of the nervous arrangements for those innumerable minute movements which fill up, so to speak, the interstices of larger movements, such as those by which we tell whether cloth is rough or not. (An explanation in all respects similar to that given, p. 275, of the spectre in some cases of Migraine is intended.)

In the third volume of these Reports, p. 337, I compared three degrees of convulsion beginning unilaterally with three degrees of the hemiplegia which is producible by destructive lesions (such as clot and softening) of different ‘gravities’ of the corpus striatum. The part of the brain destroyed is only *chiefly* motor, as the posterior part of the brain is only *chiefly* sensory (see p. 273). The degrees are arbitrary; the correct expression is that the graver the lesion (1) the more in degree are the most voluntary or special parts

affected, and (2) the further in range does the paralysis spread to the less special or less voluntary, i.e. more automatic. There is an increase of paralysis in Compound Degree. There are Degrees of Dissolution,¹ using this term as the opposite of Evolution. It will be seen that the principle of Dissolution harmonises with the principle of Compensation.

The first degree of hemiplegia (paralysis of the face, arm, and leg) is often seen after a unilaterally beginning convulsion when that convulsion has been severe. There is hemiplegia *exactly like that which a clot in the corpus striatum so often produces, or like that resulting from plugging of the middle cerebral artery*: that is to say like it in range and degree, unlike it in being transitory. This long since led me to the belief that unilaterally beginning convulsive seizures depend on disease in the region of the middle cerebral artery ('London Hospital Reports,' vol. i., 1864, and many places in 'Medical Times and Gazette'). I noticed that those parts which were first and most involved in the convulsion were the parts which were paralysed when the corpus striatum was injured by clot. I was much struck also by the confirmatory fact that, occasionally after a severe convulsion, beginning unilaterally the same parts were temporarily paralysed.²

¹ Here when for the first time in this article I use the term Dissolution, I most gratefully acknowledge my vast debt to Herbert Spencer. What I have to say of the constitution of the nervous system appears to me to be little more than illustrating his doctrine on nervous Evolution by what I may metaphorically speak of as the experiments of disease. I should make more definite acknowledgments were it not that I do not wish to mislead the reader, if, by any misunderstandings of his doctrines on my part, I impute to Mr. Spencer particular opinions he might not endorse. Anyone interested in diseases of the nervous system should carefully study Spencer's 'Psychology.'

² 'I do not assert that "epilepsy" is due to disease of [the brain in the region of] the middle cerebral artery (or of the pia mater in the range of that vessel—their vascular expansion), but I submit that one particular form of epilepsy is. Perhaps a better expression would be epileptiform convulsions,' 'London Hospital Reports, 1864,' p. 466. I now use as stated the term epilepsy for excessive, &c., discharges of any part of the cortex cerebri, and symptomatically for any symptoms thus caused. I do not make the above quotation for the purpose of claiming priority, as I find that the clinical facts of the varieties of epilepsy under remark were stated before I was born. I make the quotation because I am under the impression that it is useful for an investigator to show, as some evidence of his earnestness, that he is not putting forward hastily-considered opinions. The following disposes of any priority, I might have supposed myself to have. Charcot writes ('Revue Men-

Sometimes after a severe convulsion there is the second degree of hemiplegia (see Vol. III. p. 337), that is, there may be paralysis not only of the face, arm and leg, but in addition all lateral deviation of the eyes and head.

Here, bearing in mind the Principle of Compensation,¹ I would say that the eye and head movements escape in the first degree of hemiplegia—first degree of Dissolution—not because they are unrepresented in the destroyed units of the corpus striatum, but because they were still représentée de Medecine et de Chirurgie,' January 1877, p. 4), 'Enfin M. Hughlings Jackson s'est attaché depuis plusieurs années à démontrer que certaines lésions superficielles du cerveau peuvent déterminer une forme spéciale d'épilepsie, dont l'étude clinique avait été poussée fort loin déjà, longtemps avant, en France, par Bravais ('Recherche sur les symptômes et le traitement de l'épilepsie hémiplegique,' 1827) dans une thèse très-remarquable qui dénote chez l'auteur une sûreté de jugement et une finesse d'observation peu communes.

¹ This principle is so important that I make here further remarks on its bearing on the representation of the special senses. Because I have said that deafness never results from disease of any kind in any part of either cerebral hemispheres *so far as I have observed*, it has been inferred that I do not believe the auditory nerves to be represented in the cerebrum. Nor in my experience has disease of any kind *by its mere destroying action* in any part of either cerebral hemisphere produced blindness or defect of sight. I say by its mere destroying action, as blindness very often indeed occurs by an indirect process from disease of the cerebrum. Nevertheless I have not inferred that these nerves are unrepresented in the cerebrum. The fact is, I drew the very opposite inference from the absence of deafness and blindness from destroying lesions of the cerebrum. Thus, 'Royal London Ophthalmic Hospital Reports,' 1865, I write:—'This' (the absence of deafness in disease of the brain) 'may be due in part to the *wide connections which hearing probably has with mental functions. Probably fibres from the auditory nerve, through its nucleus, spread more uniformly than fibres of the optic nerve to the cerebral convolutions.* There can, I suppose, be little doubt that the auditory nerve does send fibres to the hemispheres directly or commissurally.'

In another part of the same paper I have written, ' . . . one would not deny that the nerve fibres of each of the special senses spread, some more and some less, directly or commissurally, to every part (although more perhaps to some parts) of *their periphery, viz., the convolutions of the cerebral hemispheres* . . . it may be that the special senses *are represented widely in each cerebral hemisphere*, and thus that much even of both the hemispheres may be destroyed without affection of sight.' The latter part of this quotation mentions expressly sight only, but implicitly all the special senses are spoken of.

I would not deny now, however, that sudden destruction to particular parts of the cerebral hemisphere may produce temporary blindness or deafness. Although I have no clinical evidence of it, I think it highly probable having regard to certain of Ferrier's experiments. It may be that I have not seen temporary blindness or deafness from sudden destruction of parts of the cerebral hemisphere, because I have not seen cases of symmetrical destruction of those parts where the auditory and optic nerves are most specially represented.

sented in every unit of the parts intact. Each unit of the corpus striatum is a corpus striatum in miniature,¹ representing slightly the whole of the parts which the entire corpus striatum represents in greater degree. But it is not supposed that each unit represents the whole of the parts in the very same way; that would be equivalent to no representation at all. The hypothesis is that the muscles of the arm, face, and leg are represented in every part of the corpus striatum, but in different combinations in each part.

Suppose the anterior units of the corpus striatum to represent movements of the arm *chiefly*, the middle units to represent those of the face *chiefly*, the posterior units those of the leg *chiefly*, the hypothesis is, that each of these groups of units represents more nearly equally with the other two the more automatic movements of the eyes and head, and still more nearly equally the movements of the thorax, and still more nearly equally other movements to be spoken of later on in this article (p. 291 and sqq.). The ocular and head movements suffer in the Second Degree of Hemiplegia, because from the graver lesions too few units are left. I submit that this is a simpler hypothesis than that which supposes these additional symptoms to be owing to extension of damage from the corpus striatum to some other centre for the eye and head movements, or than that which attributes them to 'shock.' Fully believing that the eye and head movements will be somewhat more represented in some than in other units of the corpus striatum, the great matter is gravity of lesion—the number of units destroyed and the rapidity of their destruction. The more important element in chronic cases is the number of units destroyed.

This opinion as to the constitution of the corpus striatum agrees with the Law of Dissolution (see p. 236–7), that there is from increasing gravity of lesions a twofold effect: (1), the most special or voluntary parts (arm, face and leg) are more paralysed; and (2) the further the paralysis spreads in range to less special or more automatic parts (involves movements of eyes, head, &c.). The order of Recovery confirms. The

¹ See Charcot, 'Cerebral Localisation,' p. 152. He agrees with me as to the reasonableness of this figure of speech.

more automatic parts recover first; the lateral deviations, except when a very large quantity of brain is destroyed, are transitory. Considering together the Principle of Compensation and the Law of Dissolution, it will be inferred that Recovery follows not because other units take on duties they never had before; but because these units having in health closely similar duties to those of the units destroyed, they can act nearly as well for the duties of both; it is possible that their elements, cells and fibres, hypertrophy and perhaps increase in number, in order to perform more efficiently the extra work imposed on them.

The Constitution of all nervous centres is supposed to be the same whatever their Composition may be, that is whatever parts of the body they may represent. It is just as the Form of Thought is always the same whatever may be the particular things thought about. The Principle of Dissolution and the Principle of Compensation, I suppose, hold for all nervous centres, as does also the Principle of Loss of Control; they are only different aspects of one thing. They are useful hypotheses for investigation.

In cases of post-epileptic hemiplegia, where the hemiplegia is of the Second Degree, the convulsion has been very extensive in range, and inferentially the discharge has been unusually excessive.

In a valuable paper in the 'Journal of Mental Science,' January 1876, Dr. Julius Mickle records a case of post-epileptic hemiplegia, in which conjugate deviation of the head and eyes was seen. He correctly quotes me ('Study of Convulsions, St. Andrew's Reports,' vol. iv. p. 169) as speaking doubtfully of the existence of conjugate deviation of the head and eyes in such cases. But I have since that paper was written (1870) seen cases of post-epileptic hemiplegia with those deviations.¹

Of these facts there can be no doubt whatever. That is to say, there is no doubt that hemiplegia of these two degrees follows a severe convulsion beginning in some peripheral part

¹ I may here mention that Dr. Mickle criticises an inference I drew as to the *modus operandi* of a meningeal hæmorrhage in causing unilateral convulsions. I thought then ('Medical Times and Gazette,' August 15, 1868, p. 179) that the convulsion depended on pressure on the otherwise healthy corpus striatum. I submit to the criticism. I should not nowadays draw such an inference.

of the hemiplegic region, hand, face or foot, in a patient who before that convulsion had no paralysis whatever.

I think it probable that there is sometimes, immediately after an unusually severe convulsion, and one which having begun unilaterally has *become* universal, a post-epileptic paralysis corresponding to what I believe is a third degree of, or rather a degree of paralysis *beyond* hemiplegia. (See these 'Reports,' Vol. III. p. 337.) With paralysis of the side first convulsed there is, I think, probably paresis of the side secondly convulsed.¹ The facts of 'descending wasting' show that the two sides of the body are represented in each of the corpora striata; the wasting often descends into each side of the cord, which shows that there are direct as well as decussating fibres. The conclusion from this is that the units of each of the corpora striata represent the whole of the muscles of the body, representing less those of the second than of the first side.

Respecting the principle of what was said (p. 289), the hypothesis is that the second side is still less specially represented in the units of the corpus striatum than even the movements of the head and eyes of the first side; there are fewer fibres more widely distributed, and thus it is only in gravest lesions that the second side is supposed to suffer. It can only be supposed to suffer transitorily after the severest epileptic discharges; the more automatic parts, those least and latest paralysed, recover soonest, as stated (p. 289).

Nothnagel referring to Broadbent's Hypothesis and to some previous statements of mine ('Lancet,' February 15, 1873), says, that I have *not proved* the existence of paresis of the second side; this is quite true, and as Nothnagel observes I have myself indicated those difficulties which are obvious. Let us consider the matter again after speaking of the convulsion which is supposed to correspond to the hypothetical paresis of the 'second side.'²

¹ It is convenient in what follows to use the expressions, 'first side' and 'second side.' Thus, with reference to the right corpus striatum the left side of the body is the first side, the right side the second side. This is the physiological order of the representation of the two sides in the right corpus striatum.

² Nothnagel states that Broadbent believes the two halves of the body to be represented in each cerebral hemisphere. So far as I know Broadbent does not

On the doctrine of evolution the centres next higher (those in Hitzig and Ferrier's region) than the corpus striatum will each of them represent again, but in greater complexity and speciality, all the parts the corpus striatum (and doubtless centres still lower, geographically), has already represented. Yet each of the higher centres in Hitzig and Ferrier's region is supposed to represent more specially some particular part (face, arm or leg), and each more generally all other parts, than they have been represented in the corpus striatum. We call these centres which are higher in degree of evolution than the corpus striatum, subordinate motor centres, because they are lower in evolution than the centres which are the substrata of consciousness. Let us call them middle centres. Each of these middle centres will re-represent movements of both sides of the body, because each of them represents the whole of the parts already represented in the corpus striatum.

If this be the constitution of the middle centres, there is, I submit, no difficulty in understanding how it is that in epileptic discharges of *one* of them, or discharge beginning in one of them, the convulsion after affecting one side of the body, the first side, the currents passing by the route of the decussating fibres, should become universal—that the spasm should 'travel' to the other, the second, side of the body, the currents passing by the route of the direct fibres. It is fair to say that so far as I know, I am alone in this opinion as to the mode of causation of convulsion of the second side. Nothnagel says that the convulsion of the 'second side' may, in his opinion, be explained quite as well by the assumption of a secondary sympathy of the other cerebral hemisphere, or by a secondary affection of the pons or medulla oblongata.

It is important to note next that the two sets of wasted fibres 'descend' into different columns of the cord; into the lateral column of the opposite side (first side), and into the

suppose, as I do, that muscles of the arms and legs are thus doubly represented. I make this remark, as I do not wish to impute to him views he may not share with me. I do not make the remark because I wish to claim any priority. I have written elsewhere ('Medical Press and Circular,' September 6, 1876), 'I have carried the hypothesis further than Broadbent, but subject to his approval, I should say that I have added *nothing* to the *principle* of that hypothesis.'

anterior column of the same side (second side); the wasting in the lateral column affecting that part of it where there are most fibres of smallest diameter; the part of the anterior column being that where there are the thickest fibres of that column—the largest of all in the cord. This difference of necessity implies differences of *kind* of representation of the corresponding muscles of two sides of the body in each half of the brain if the fibres of the anterior column be the route of the current for the ‘second side,’ for we cannot suppose the anterior columns to have just the same duty as the lateral columns.

We should expect, then, that when a fit begins in the hand of the ‘first side,’ that the spasm in the convulsion of the ‘second side’ would invade the parts of that second side in a manner different from the invasion of the first side. There are, however, very few facts as to the mode of convulsion of the second side. My colleague, Dr. Gowers, has made some important observations on this matter. He writes (*‘Lancet,’* November 6, 1875) of one case: ‘Invariably one arm (right) and both legs were convulsed, the other arm escaping entirely. . . .’¹ It is to be observed that in the right leg spasm of the extensors predominated, while in the left leg [‘second side’] that of the flexors was distinctly greater than that of the extensors.’ The two legs, however, were convulsed simultaneously.

From a few observations, and from hypothetical considerations, I should expect that when a convulsion begins in the hand the spasm reaching the ‘second side’ would affect first and most the parts affected last and least on the first side (head, eyes, leg, trunk first and limbs last). Hence, believing the paresis to be the counterpart of the spasm—not equal in range, however (see p. 286), I should not expect it to affect most those parts which on the undoubtedly paralysed side are most affected. I should expect it on each side to affect those parts most which have been first and most convulsed on that side. I do not pretend to have demonstrated that any paresis occurs on the second side; we should expect

¹ The part omitted in the quotation is, ‘May it be connected with the more frequent simultaneous use of the legs than of the arms?’

the paresis to be transitory. For if the Law of Recovery holds (see p. 289) here, any paresis there was would be transitory, and might disappear before the patient had regained full consciousness. (See p. 305, on loss of power during loss of consciousness and coma.)

I expect then, that if there be paralysis of the second side it will be slight and transitory, and that it will not be of those parts *most* which are *most* affected in the paralysis of the first side.

That several of the above statements are hypothetical is no objection. Hypothesis is necessary in the investigation of complex and difficult subjects; an hypothesis is not a conclusion; it is only a provisional conclusion. But that my hypothesis that there is post-epileptic paresis of the second side is one about which I can only slightly indicate methods of investigation for proof or disproof, is almost an overwhelming objection. There is, however, I think further, although indirect, evidence to be adduced.

It is convenient to adduce this evidence for another reason, viz. in order to enforce what was said (p. 272) as to the Constitution of the substrata of consciousness—that they represent or re-represent the impressions and movements of the whole body. Surely this is evident *à priori*; since consciousness represents the whole Subject,¹ must not its substrata represent the whole Organism?

We shall approach the statement of this evidence by carrying still further the hypothesis of nervous evolution, and we shall see, I think, that the evidence when stated entirely agrees with this hypothesis, although it is quite independent of it. We continue to illustrate by the motor element only, except in a few preliminary remarks.

It is not of course supposed that the substrata of consciousness are motor only, nor that they are in but one side of the brain. It is supposed that each cerebral hemisphere represents the impressions and movements of the whole

¹ We should say Subject-Object, for the correspondence of the organism with the environment is duplex. There are highest nervous arrangements by which we have received and are continually receiving impressions from the environment (substrata of subject consciousness, chiefly sensory), and highest nervous arrangements by which we can and do react on the environment (substrata of object-consciousness, chiefly motor). *

organism — the anterior part of each hemisphere being chiefly motor, the posterior chiefly sensory (see pp. 273–5). There is I think the broad distinction that the left posterior part and the right anterior part are the substrata of Subject-consciousness, that is to say, they represent the whole organism as it is affected by the environment—and that the right posterior and left anterior parts are the substrata of Object-consciousness, that is to say, they represent the whole organism as it reacts on the environment. Let us speak of sensations; we must not however narrow ourselves to the sensations of the special senses; there are what Lewes calls ‘systemic,’ and Bain ‘organic’ sensations. A complete study of the senses under this enlarged definition would be a study of the whole body. All the senses are supposed to be sensori-motor: every sensation occurs during energising of sensori-motor arrangements. Still as disease to some degree can separate the two elements (see p. 275) we shall speak of motor-sensations and sensory-sensations; these being brief expressions for ‘sensations occurring *during* energising of sensory and motor elements of nervous arrangements.’

I suppose the organic sensory-sensations from tissues, viscera, muscles, &c., to have first and most representation in the posterior part of the left cerebral hemisphere, and the visual, auditory, &c., sensory-sensations, the first and most representation in the posterior part of the right: again, that organic-motor sensations would be first and most represented in the anterior part of the right; the visual motor-sensations first and most in the anterior part of the left. This is equivalent to the statements already made as to seats of the substrata of Subject and Object consciousness.

Let us see how this hypothesis as to the particular constitution of the several divisions of the highest centres bears on the investigation of epileptic paroxysms. There are warnings by morbid development of sensations. When there are warnings of course the epileptic discharge does not begin in the very highest arrangements of the highest centres. But many of these warnings are very quickly followed by loss of consciousness, and thus loss of consciousness being

almost the first thing, the discharge must begin in nervous arrangements which are next to the highest in the highest centres. At p. 270 we gave a rough list of warning-sensations. There is the strange feeling at or near to the epigastrium so very common just before complete loss of consciousness; this is, I think, a development of highly complex systemic sensory-sensations. There is eructation or nausea (physically incipient vomiting), a development of systemic motor-sensations. Again we have the sensory-sensation of colour, and the motor-sensation of vertigo. It is important to note all warnings by different sensations, and with regard to accompanying physical manifestations. It is quite as important to note that a patient has the organic motor-sensation of shivering as a warning of his paroxysm as that a patient has that of coloured vision. And in order to see whether or not different sensations have the order of degree of representation in the several parts of the cerebral hemisphere we have suggested we should note carefully the convulsion which follows each. Thus after any particular sensation-warning do the two eyes turn to the right or to the left; does the convulsion begin in or preponderate on the right or left side? Such facts would tell us whether or not the sensation developed as a warning was owing to discharge of the right or left cerebral hemisphere. It is a strange thing that from epileptic discharges, which we judge to begin nearly in the highest nervous arrangements of the highest cerebral centres we have sensations at both ends of the scale; we have the most special, as coloured vision,¹ and the least special, as shivering. The explanation is, I think, that the substrata of consciousness are (p. 295) duplex in function. All centres are bilateral, but the higher the centres, the less of a mere duplicate are they

¹ I believe, however, that the more special the sensation the more unilateral is the convulsion, if there be one.

As stated on p. 274, I believe cases of Migraine to be epilepsies (sensory epilepsies). Dr. Latham thinks the paroxysm in Migraine to be owing to arterial contraction in the region of the posterior cerebral artery; Dr. Lieving that there is a 'nerve storm' traversing the optic thalamus and other centres. I think the sensory symptoms of the paroxysm are owing to a 'discharging lesion' of convolutions evolved out of the optic thalamus, i.e. of 'sensory middle centres' analogous to the 'motor middle centres' (see p. 292). I believe the headache and vomiting to be post-paroxysmal.

in function on the two sides; in the highest centres this difference in the doubles is carried to an extreme; the substrata of Subject-consciousness representing the organism as a whole, and as it suffers from the environment and the substrata of Object-consciousness representing the whole organism as reacting on the environment. The more quickly consciousness is lost after a warning, the more often is that warning low in the scale of sensations, 'epigastric aura,' &c.

I do not mean that the above statements are true; they are only rough hypotheses for verification or disproof.

The occasional occurrence of coloured vision at the onset of some *right-side* beginning convulsive paroxysms (as was the case in Dr. Robertson's patient, p. 285) shows that these differences of the two sides of the brain are not so simple as they are here hypothetically stated. I have recorded a case of a patient who was subject to convulsion beginning in the right thumb, and who *at other times* had attacks of coloured vision ('Medical Times and Gazette,' June 6, 1863). Still I believe that most commonly warnings of coloured vision, noises in the ear, &c., are owing to discharging lesions in the right cerebral hemisphere.

Let us now consider the effects of epileptic discharge of motor elements of the substrata of consciousness; the discharging lesions being in but one side of the brain. We now apply the hypothesis of Evolution to the highest centres, the substrata of consciousness, speaking only of the motor elements.

The very highest motor centres are supposed to be evolved out of the subordinate or middle cerebral centres in Hitzig and Ferrier's Region, and perhaps out of still lower centres by fibres not passing through the central ganglia of the brain. Thus the highest centres differ from the middle centres only as the latter differ from their lower centres, (*corpus striatum*, &c., see p. 292); the principle of difference being in each case a re-representation of the very same external parts, in more special and complex ways.¹

¹ I say the *principle* of difference; there is, however, an obvious qualification. The higher a centre, not only the more complex is its re-representation of the parts lower centres have represented, but there is in many cases also an introduction of elements for new impressions and movements.

The corpus striatum represents movements of both sides of the body; these are re-represented in the middle cerebral motor centres, and they are re-re-represented in the highest cerebral motor centres—motor division of substrata of consciousness.

Thus units of these highest motor centres of each side of the brain represent the most special and most complex movements of the whole body, or rather of the body as a whole.¹

It is not of course supposed that any unit represents the whole body—or a large part of the body—so that all the parts it represents are equally represented. The kind of representation is supposed to be analogous to the kind of representation attributed to middle centres in Hitzig and Ferrier's Region. Each unit of the highest centre represents some part of the body very specially, and all other or very many other parts very generally, *i.e.* in common with, more equally with, many other units.

The units of the highest centres represent the whole body in innumerable different combinations of impressions and movements. 'The seat of consciousness,' Herbert Spencer says, 'is that nervous centre to which the most heterogeneous impressions are brought,' and I suppose that he would hold also that it is that centre from which impulses for the most heterogeneous movements depart. There is in the highest centres—the centres of centres—a most intricate and yet orderly Space and Time co-ordination. There is not only a representation of a great number of different movements of widely separated parts of the body, but a representation of these at a great number of different intervals.

Thus far we have simply been carrying out the hypothesis of Evolution. Now let us consider the experiments disease makes on these highest centres.

The Epileptic discharge of many units of these highest

¹ An instance can be adduced showing that a very small part of the organism represents the whole organism, the sperm cell and germ cell; these united grow into a complete organism without any other help than the supply of warmth and nutrient material in a woman's womb. Were I to adopt any doctrine of genesis it would be that the germ and sperm cells are made up of detachments from the highest nervous centres.

centres puts in action both sides of the body ; that is to say, the convulsion in those cases in which there is loss of consciousness at the outset of the epileptic paroxysm is bilateral. However, it is not supposed that even in the very highest centres the movements of the two sides of the body are represented quite equally in each side of the brain. It is only supposed that the two sides are represented more nearly equally than in the middle centres. The bilateral convulsion, in cases beginning with loss of consciousness, commonly begins by turning of the eyes and head to one side, and the universal convulsion usually preponderates on one side. All that is said is that the two sides are more nearly equally convulsed in epileptic discharges of the highest centres than in discharges of middle centres. The several parts are convulsed more nearly contemporaneously. For, as stated, the innumerable different movements are represented in innumerable different intervals—so to speak, the time is divided into very numerous parts. Moreover, corresponding partly, I suppose, to differences in the sizes of the nerve cells, the epileptic discharges of the highest centres containing more small cells are more sudden and rapid, more ‘intense’ (great quantity of energy liberated in a short time), than epileptic discharges of the subordinate or middle cerebral centres in Hitzig and Ferrier’s Region, where are many large cells. To resume, the convulsion is more quickly universal, the two sides of the body are more nearly equally and more nearly contemporaneously convulsed when part of the highest centres is discharged, than when a middle or subordinate cerebral centre is discharged.

The convulsions from epileptic discharges of middle centres in Hitzig and Ferrier’s region can now be more clearly compared with convulsions from discharges of the highest centres. I make this comparison because I have been misunderstood to believe that all epilepsies are owing to discharging lesions in Hitzig and Ferrier’s Region. I never believed the ‘genuine’ epilepsy of authorities—that is, epilepsy beginning with loss of consciousness—to be owing to discharging lesions in this region. (See footnote 2, p. 287.) The following quotation, in which some verbal

alterations are made, referring to the convulsions of 'genuine' epilepsy and to unilaterally-beginning convulsions, shows that in 1870 I thought as I now think as to the differences betwixt the seat of the lesion in each:—

'Those who say that the two classes differ "only in degree" make a remark the truth of which is admitted. In both there are occasional, excessive, and disorderly expenditure of *energy* on muscles, the discharge depending on instability of nervous tissue. But in what kind of degree do they differ? Not merely in degree of more or less spasm—more or less instability of nervous tissue—but also *in degree of evolution* of the nervous processes which are unstable. A convulsion which is *universal*, and in which the muscular regions affected are affected nearly contemporaneously, must depend on discharge of parts in which the nervous processes represent a more intricate co-ordination of *movements* in Space and in Time than those parts represent which, when discharged, produce a convulsion, which begins in one limb and has a deliberate march. My speculation is, that the first class differs from the second in that convulsions at a greater physiological distance from the motor tract are discharged.'¹

It may, perhaps, be said that this hypothesis as to the constitution of the motor part of the substrata of consciousness is founded on the hypothesis stated (p. 294), where it was also said that an hypothesis is not a conclusion. But it was said, too, that an hypothesis is a provisional conclusion; and it is quite legitimate to carry further, as we have done, the one stated, to see if it will explain the facts we observe. But the statements as to the constitution of the highest centres rests not on that hypothesis, but on quite another kind of evidence, which agrees with it. The paroxysms just spoken of are those which *begin with* loss of consciousness; and this is equivalent to saying that the epileptic discharge begins in the highest centres. The effect of an epileptic discharg-

¹ This paragraph is reproduced from the 'Study of Convulsions,' 1870, with the alterations of 'force' to 'energy,' 'general' to 'universal,' and 'muscles' to 'movements,' and with the intercalation of the word 'physiological' in the last line but one.

ing lesion there seated is convulsion of both sides of the body; the two are more nearly equally and more nearly contemporaneously convulsed than when the discharging lesion is of middle or subordinate centres. The convulsions in 'genuine' epilepsy may be looked on as experiments made by disease (using this expression metaphorically), which reveal to us the nature of the constitution of the highest centres (their motor elements). The evidence supplied by these cases is the 'indirect evidence' spoken of at p. 294.

Of course there will be degrees betwixt the two classes of epilepsy. For among those epilepsies which are commonly classed as genuine epilepsy there are cases in which, whilst loss of consciousness is almost the first thing, there is yet some premonitory sensation (vertigo, shivering, creeping, &c.), showing that the discharge does not begin in the very highest arrangements of the highest centres, or consciousness would be lost at the very first. We should theoretically expect there to be *all* degrees betwixt the two great classes, and possibly there are. But it does not follow that there will occur at any rate *as numerous* intermediate degrees as are theoretically likely; for we have to take note that some parts of the nervous system are more liable to *become diseased* than others are: it is evidently so as to clot or softening; the peculiarities in the distribution of arteries account for the 'seats of election' of these two morbid conditions even in details, as Duret has shown. It is, indeed, a very singular thing that there do occur the two groups of epilepsies with few intermediate cases showing in their paroxysms an intermediateness of degrees of bilaterality, and of degrees of contemporaneity of convulsion. I suppose these differences will be accounted for by differences in arterial distribution.

But it may be asked, as was anticipated (p. 269), why is consciousness lost when there is a discharging lesion of but *one* part of *one side* of the brain? Currents developed by discharging lesions will pass 'laterally' as well as 'downwards'—will pass to associated centres of the same rank, so to speak, as well as to lower centres.

The higher the centre the more numerous different im-

pressions and movements it represents. But this is not all. Along with increasing division of labour there goes increasing co-operation of labourers; increasing nervous differentiation is attended or is followed closely by more complete integration; the higher the centre, the more 'lateral' connexions betwixt the nervous arrangements of the same hemisphere and of the two hemispheres. It is evident *à priori* that the highest centres must be more integrated than the lower; their unity is chiefly the unity of co-operation; the unity of lower centres is more the unity of undifferentiation. Hence the units of the very highest centres which represent the most numerous different movements and impressions will have most interconnexions. We see, indeed, in the scale of animal life that the corpus callosum is more and more developed the more independent are the movements of the arms: in the bird whose arms (wings) act equally in range and together in time there is no corpus callosum.

Thus the currents from a discharge in but one of the divisions of the highest nervous centres (see p. 295) will not only pass 'downwards' but will pass 'laterally' to other divisions of the highest centres with which the centre discharged is in physiological connexion; thus the loss of consciousness from a local discharge is accounted for; the currents developed will affect the brain widely. In some cases (*le petit mal*) of epileptic discharge of the very highest centres the 'lateral' currents are chief; there is thoroughly complete loss of consciousness with little visible peripheral change; that is to say, little evidence of currents passing 'downwards' (see p. 271); in some cases there is scarcely any, practically no, outward manifestations. When so the loss of consciousness is often most transitory. It is well known that some epileptics lose consciousness absolutely, and yet for so short a time that strangers sitting opposite them at dinner may notice no change. So much for local discharging lesions of the highest nervous centres.

It is otherwise with destroying lesions; it is very true that at the time the lesion is effected, if it be sudden, as hæmorrhage, there is 'shock' doubtless from sudden development of wide-spreading currents by rapid tearing up of

grey matter ; these are effects of the mere act of destruction ; there may even be convulsion. But from the mere absence of part of one of the highest divisions of the substrata of consciousness there is no *loss* of consciousness, for the part of the centre undamaged and the other divisions of the substrata of consciousness remain unaffected.¹

In discharging lesions of the highest centres there are repeated discharges, and thus much of the brain will be disturbed again and again.

In this connexion it is convenient to note that the epilepsies of which the discharging lesions are seated in the highest nervous arrangements are those which occur in asylums. Clinically speaking asylum cases are most often those in which the paroxysms begin by loss of consciousness, which is equivalent to the anatomico-physiological statement that the discharge begins in the highest nervous centres. In equivalent clinical language we say that asylum cases of epilepsy are often those which begin without a warning. Crichton-Browne says (these Reports, Vol. III. p. 160) : ‘ In lunatic asylum practice an aura is of rare occurrence in epilepsy.’ He says that, using the term aura in its widest sense to include ‘ all prodromata, all symptoms, psychical, sensory, or motor, which immediately precede a paroxysm, an aura is rare in a lunatic hospital.’ He says, however, that the absence of aura may be owing to the fact that the patients are often far advanced in the disease when admitted ; and again it may not be recognised by the patients because their feelings and intelligence are blunted. But in cases which occur in asylums there is sometimes an aura, but loss of consciousness soon follows ; loss of consciousness *is almost* the first thing. Then the warnings are of a particular kind. They are frequently vertigo, epigastric sensations, or a universal creeping, tingling, or thrilling of the skin. On this matter I again quote Crichton-Browne. In a letter to me in reply to enquiries he says : ‘ When auras are present in epileptic lunatics they are almost invariably general in character and consist in indescribable feelings in the head

¹ For a long time ; general cerebral atrophy follows on local damage to the brain.

(vertigo) or abdomen, spreading thence over the body, or in universal creeping, tingling, or thrilling of the skin. Of course special warnings are occasionally encountered, but in a great majority of cases there is no warning of any kind.' Thus the 'auras' lunatic epileptics have are (1) most frequently those which imply developments of the most highly representative of all movements (vertigo on its physical side is a discharge of ocular motor centres); and (2) those which there is good reason to believe imply developments of the most special of Systemic sensations (the epigastric aura).

Again, the very cases in which the intellect is most rapidly affected are those in which there are very slight attacks beginning with or apparently constituted solely by loss of consciousness—*le vertige épileptique* and *le petit mal* of the French. But this is not because the attacks are slight; it is because the 'disease' is of the very highest nervous arrangements in the whole nervous system, and of those which have the greatest integration, that is to say, of the substrata of consciousness. The following quotations are given to enforce this statement:—

'The greatest injury to the intellect is not inflicted by the most frightful and frequent convulsions, nor when the mature and muscular man struggles like a chain-bound Hercules. Absence of mind, momentary obliviousness, vertiginous feelings, a pause, a stoppage, an intermission in consciousness, such as has been described as the *petit mal*, as surely and swiftly produce enfeeblement. This was the conviction of Esquirol. It has been corroborated by many of equal discrimination.'—*Epileptics: their Mental Condition*, by W. A. F. Browne, 'Jour. of Mental Science,' October 1865.

'On a remarqué que l'absence, malgré sa légèreté apparente est la forme la plus redoutable au point de vue de l'altération des facultés intellectuelles.'—*Jaccoud*, 'Pathologie Interne,' p. 394.

Is there a condition after the convulsion from discharge beginning in the highest centres (the substrata of consciousness), that is to say, after the convulsion in those cases of epilepsy which *begin with* loss of consciousness, which compares with post-epileptic palsy of a limb? I think there is

a universal or widespread diminution of power corresponding to the universal convulsion. There is no local palsy anywhere demonstrable; the loss of power is universally distributed, as the spasm in the convulsion was.

It may, however, be said that the universal loss or weakness of movement is simply owing to the patient being comatose—that it is apparent only. But what is it for a patient to be comatose? Here, again, from another standpoint we must distinguish betwixt psychical and physical. First, then, for the psychical side of the condition. To be comatose is only a greater degree or greater depth of loss of consciousness, as this is only a greater degree of that slight defect permitting only slight confusion of thought (defect of consciousness). What are the physical states corresponding to these psychical losses? Of course there must be negative states of nervous centres in these cases, as certainly as there are positive states during degrees of consciousness.

There is no such entity as consciousness; in health we are from moment to moment differently conscious. Consciousness varies in kind and degree according as the parts of the brain in activity are different, and according to the degree of their activity; and it varies in depth. Object-consciousness is continually changing and varying; Subject-consciousness is comparatively persistent and unvarying.

The three degrees we have taken for illustration are of course arbitrary; every physician knows that there occur all degrees, from the slightest and most transient defect of consciousness (attended by slight confusion of thought) to coma, and below this to death, psychical and physical. The patient who has slight defect of consciousness has lost the use of a part only of the very highest nervous arrangements; the patient who has lost consciousness has lost more of them; the patient who is comatose has lost still more. And as the highest nervous arrangements are only highly complex sensori-motor arrangements representing the whole organism, there are degrees of loss of power over the body corresponding to what are psychically degrees of affection of consciousness. In the case of a man who is comatose after an epileptic

fit there will of course be more than this; not only will the highest centres be exhausted, but more or less of many lower centres through which the currents developed have passed.

To say that a man staggers or lies motionless *because* he has lost consciousness or is comatose, is like saying that a man does not speak *because* he has lost the memory of words, which, at the best, is only repeating in technical terms that he does not speak; it is like saying that a man does not move his arm *because* he has lost volition, which is only repeating in technical terms that he cannot move it. Nobody is any the wiser for these 'explanations'; they explain nothing at all; they are attempts to explain physical states by mental states. They may be convenient empirically, but scientifically they are misleading.

Many talk as if a patient could lose consciousness without a loss of use of some parts of the nervous system; or if they admit that there is loss of use of nervous arrangements along with loss (psychical) of consciousness, the inference from the statement of that admission often is that the nervous arrangements affected did not represent parts of the body, but are centres having nothing to do but to 'play upon' the lower centres; morphologically they are spoken of as part of the body, but physiologically they are spoken of as if they were as distinct from it as the most psychological of psychologists supposes mind to be independent of organisation. This view of the matter, indeed, only differs from that of those medical psychologists who speak of an immaterial mind influencing the body, in that the mind is considered to be solid. Those who take this view are not materialists in effect, nor psychologists either, but a little of both. On this view the centres for consciousness are not 'evolved out of,' but 'placed upon,' lower centres and 'govern' them autocratically. There is no practical difference in a medical enquiry betwixt this view and that of those psychologists who speak of will, memory, &c. as 'faculties' existing apart from physical organisation. On this metaphysico-materialistic view, which really ignores anatomy, substituting for it mere morphology, the centres for consciousness are

not centres for receiving impressions, and giving out impulses for movements, but centres which 'play upon' lower centres for movements autocratically. On this view, if the centres for consciousness could be sliced away without disturbing the rest of the organism, the physical operations of the body would be positively uninterfered with, although negatively there would be no 'volitional impulses' sent down to put the body in this or that movement. Such is, indeed, the explanation actually given by some of the effects of removal of the cerebral hemispheres in certain lower animals. It removes the animal's mind, but does not affect its movements. The view taken in this article is an anatomical one, viz., that the substrata of consciousness are sensori-motor arrangements re-representing all lower centres, and thus representing the whole organism. They govern it not autocratically, but *because* they represent it, and represent it in a far higher degree than an elected President represents a republican state. They *are* the organism in potentiality, giving out impulses to most heterogeneous movements, because they receive most heterogeneous impressions. On this hypothesis if they were sliced away there would be loss of innumerable highly complex and most special co-ordinations of the body as a whole, the lower co-ordinations being badly effected, not from want of autocratic governance, but from actual lack of the higher co-ordinations; there would be a degree of paralysis universally distributed. Volitional impulses are in this view only the psychical side of activity of the very highest sensori-motor arrangements respecting the whole body. The animal who from lack of the experimentally removed cerebrum has no volition has lost those highest sensori-motor arrangements during activity of which automatic mental action breaks down into volitional action; or, speaking of the physical side, where the most complex reflex action becomes imperfect and ceases.

The explanation here combated is a metaphysical one. It is a very common thing in medical writings to meet with pure metaphysics put forward as if they were something highly practical. Let me illustrate this statement by an

example. In disease of the cerebellum a person is said to reel *because* he has lost the use of a co-ordinating centre. This need not be cavilled at. But then, unfortunately, the supposition often is that there *is* a co-ordinating centre which does not itself represent the most elaborate combinations of impressions and movements of the parts to be co-ordinated, but as it were stands outside or above the centres which are for impressions and movements and autocratically governs them, regulates them.

But the man who reels slightly from slight disease of the cerebellum does so because he has lost power over the muscles of his spine; has actually *lost* the most special and most delicate nervous arrangements for balancing movements of the trunk, those which occur first in locomotion. Here again we see a duplex symptomatic condition. (See foot-notes at pages 271 and 285.) The movements the patient *does make* are owing to action of healthy centres; the legs are doing their duty faithfully in running after the trunk to prop it up in its various over-inclinings. Not seeing that there are two elements (negative and positive) leads to the ignoring of the fact that the patient has *lost* something (negative) which is the indirect cause of the over-action of his legs (positive).

We must observe that in each of the three degrees of affection of consciousness (see p. 305) the condition is duplex. In the first degree (defect of consciousness) there is positively confusion of ideas, with the third there is positively conservation of such deeply automatic movements as those of respiration. For example, the confusion of ideas is owing to want of co-operation of those highest nervous arrangements whose loss is attended by the slight psychical affection we call defect of consciousness; even confused ideation implies activity of nervous arrangements.

Here ends the general sketch, and here too must end my Paper, although I have not yet adduced many important facts which, I think, strongly support the statements made. In particular I have not stated the facts supplied by very limited palsies after very limited convulsions—as of an arm

after convulsion of little more than that limb. The reader is asked to bear in mind that this is only part of a Paper, and that therefore the argument is incomplete. The remainder of the Paper will, if my engagements permit, be printed for private circulation with a reprint of the foregoing.

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